VOLUME 5 FEBRUARY, 1932 NUMBER 8
(Old Series, Vol. X)

ANNALS OF INTERNAL MEDICINE

PUBLISHED BY

The American College of Physicians

CONTENTS

Issued Monthly
Ann Arbor, Michigan

Sixteenth Annual Clinical Session, San Francisco, California, April 4-8, 1932

ANNOUNCING—a new kind of Medical History—so entertainingly written that you will always treasure it, yet a veritable storehouse of information.

HISTORY *of* MEDICINE *in* THE UNITED STATES

By FRANCIS R. PACKARD, M.D.
Editor of Annals of Medical History

EVERY physician and surgeon, in fact, every cultured person, will find this new work extremely worthwhile. While it is a veritable storehouse of information, tracing the development of the history of medicine in the United States, it is more than a chronological presentation of historical facts for it reads as easily as a novel.

Accounts of the Doctors' Mob, the Controversy over Inoculation, the Establishment of the Revolutionary Hospitals, the Various Disputes over the Location of Medical Schools, and hundreds of other events of more than usual interest are here presented in interesting style and enriched by intimate accounts of the personalities of the various periods.

This is not an ordinary history of medicine, for it is embellished with details gathered from many sources and written so entertainingly that one is loath to put the book aside.

TWO VOLUMES, 8VO, CLOTH, 1348 PAGES, 103 ILLUS. $12.00 NET

PAUL B · HOEBER · INC · PUBLISHERS
SEVENTY-SIX FIFTH AVENUE · NEW YORK

Publishers of *Annals of Medical History; The American Journal of Surgery; Clio Medica; Annals of Roentgenology, etc.*

Multiple Arterial Occlusions*

By Harold W. Dana, M.D., F.A.C.P., *Boston, Mass.*

FOR many years we have been cautioned, with regard to the use of quinidine in the treatment of auricular fibrillation, of the possible danger that we might cause the extrusion into the circulation, when the previously dilated auricles should contract, of thrombi formed in these inactive chambers of the heart. Such an accident would seem not only to be possible, but even to be likely, should any method of treatment be successful in re-establishing rhythmic contraction of auricles long inactive, and undoubtedly this misfortune does at times follow the use of this drug, although much less frequently than one would expect upon theoretical grounds.

Outside of the cinchona group, no drug is known to have an effect on auricular contraction. Digitalis, though widely used in fibrillation, is employed only as a means of setting up a barrier against ventricular overstimulation. Mackenzie[1] in speaking of the danger from the use of digitalis in fibrillation, was referring only to the possibility of producing too great a degree of block after the pulse rate had become sufficiently slow. Hare[2] refers to the possible dangers of massive doses of digitalis in patients with a tendency to embolism "arising in all probability from the valvular disease", apparently quite apart from any discussion of the effect of this drug in fibrillation.

In this paper I should like to present the hypothesis that pituitrin, either alone or in conjunction with adrenalin or digitalis, may be capable of causing the stationary auricles to contract rhythmically. The evidence is based upon the apparent effect of one or more of these drugs in causing what I believe to have been multiple embolism, with the simultaneous disappearance of a pre-existing fibrillation of the auricles. The patient in whom this occurred was, on this particular admission to hospital, under treatment for lobar pneumonia, and after having been digitalized, she had received repeated subcutaneous injections of pituitrin because of circulatory failure as evidenced by falling systolic and pulse pressures. She had also received one or more injections of adrenalin during one, at least, of the two severe chills with partial collapse following each of two large intravenous injections of glucose. There was no evidence of an endocarditis. No quinine had been used in treatment.

The effect of pituitary extract upon unstriped muscle, in the uterus, the intestine, and the blood vessels, is long established; within recent years

*Submitted for publication, June 9, 1931.

several observers[3,4] have noted the emptying of the gall bladder as the result of the action of this agent; and since cardiac muscle is closely related to unstriped involuntary muscle, it would seem at least theoretically possible to obtain some contractile influence upon the heart from pituitrin, which Dale[5] believes does in fact occur. Twenty years ago, however, Wiggers,[6] in a series of experiments both on the perfused heart and by intravenous injection, found that pituitrin did not cause any increase in the amplitude of cardiac contraction. More recent experimenters, as Ross with others[7], Kolls and Geiling[8], Geiling and Resnik[9], speak of the possibility of direct action of the extract on heart muscle, or of indirect action through coronary constriction, though rather in causing dilatation than increased contraction.

It would seem that the only possible explanations for what occurred in the patient referred to, are as follows:

(1) that the fibrillation was due to the pneumonia and disappeared when the patient's condition improved;

(2) that there was a spontaneous cessation of a paroxysmal fibrillation, and

(a) that emboli were thereby extruded into the circulation, or

(b) that thromboses occurred in various locations, also spontaneously, or

(c) that the thromboses were due to the effect of pituitrin locally in causing narrowing of the capillary bed in the extremities;

(3) that both the return to normal auricular rhythm and the occurrence of embolism were due either to pituitrin or to its effect combined with that of other drugs. Whether such possible effect of pituitrin might have been through stimulating the contraction of the auricles or through its depressant effect upon cardiac muscle, or by stimulation of the cardio-inhibitory center, will not be discussed.

Why glucose injections, when given in pneumonia, should produce, at times, a condition simulating anaphyiactic shock, has not as yet been satisfactorily explained. It is possible that such a reaction may be due to massive destruction of leucocytes with the production of toxic substances. That either glucose shock or the single injection of adrenalin used in its treatment could have caused thrombosis in this case does not seem likely, since there was an interval of six days between the last use of these agents and the appearance of any arterial occlusion.

That fibrillation may have resulted from the pneumonia has no particular bearing, since there had been undoubted previous attacks of fibrillation. In fact, there is no actual proof that a spontaneous termination of fibrillation did not occur in this case. While the patient had had "palpitation of the heart" for eight or ten years and definite symptoms of cardiac decompensation for three years, when admitted to hospital one year ago with a diagnosis of auricular fibrillation, the heart action was reported as regular in all examinations during her three

weeks' stay, and no fibrillation was diagnosed at that time. However, no electrocardiographic examination was then made, for some reason, so that fibrillation with regular ventricular action cannot be positively excluded.

If, during the patient's latest stay in hospital, there had been a spontaneous restoration to normal rhythm, then the fibrillation can be assumed to have been paroxysmal in type, of short duration, unlikely to have produced clot formation and extrusion of emboli.

With regard to the possibility that there could have been a spontaneous re-initiation of auricular dynamic contraction, with an almost simultaneous onset of widely scattered thrombus formation due to the local action of pituitrin upon the blood vessels, there are the following difficulties to be explained. In the first place, this requires belief in a most remarkable coincidence; further, the vessels involved in the hands and feet were not merely capillaries, but vessels of good size; again, the first evidence of the process occurred in the tip of the nose, then in both feet, next in the right hand, later on the left forearm, probably in the lungs, and finally on one cheek, this distribution of lesions being spread over several days; and as a final argument, the process in the cheek seemed rather definitely embolic, in that there was pus formation.

Unfortunately, the evidence in the case is not at all complete; for various reasons, chiefly mechanical, no electrocardiogram was taken until after the heart action had become regular, and eight days after the first evidence

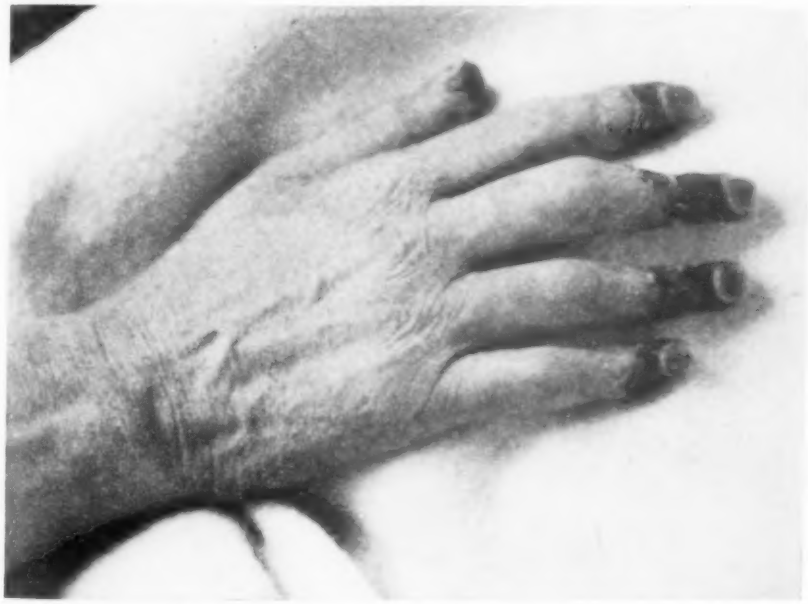

FIG. 1. Photograph of right hand, taken Feb. 15th, 1931, showing condition of fingers at the time of discharge from hospital.

of interference with peripheral circulation. Further, while it is the impression of several of us that the return to normal rhythm occurred at the same time that onset of gangrene of the extremities appeared, this cannot be substantiated from the hospital records.

Very much to the point is the observation of Halsey[10] in Barker's Endocrinology. Rénon and Delille are quoted by Halsey as stating that arrhythmias were not influenced by pituitrin, but that in several cases tachycardia was controlled after other therapy had failed; and to this Halsey makes the commentary, "Every clinician of sufficient experience is aware of the difficulty of determining whether the cessation of one of these paroxysms following any line of treatment is an instance of *post hoc* or *propter hoc*. However, there is pharmacological evidence that the injection of extracts of this gland may directly depress cardiac muscle and in this way it is possible that such would exert a favorable influence in tachycardia."

Resnik and Geiling[11] made an electrocardiographic study of the action of pituitary extract and showed changes in the heart rate, partly due to stimulation of the cardio-inhibitory centre and partly to a "muscular action". It is possible that the low amplitude of all waves in the earliest electrocardiogram from the case here reported is due to the depressant action of pituitrin, rather than to any specific action in making the auricles contract.

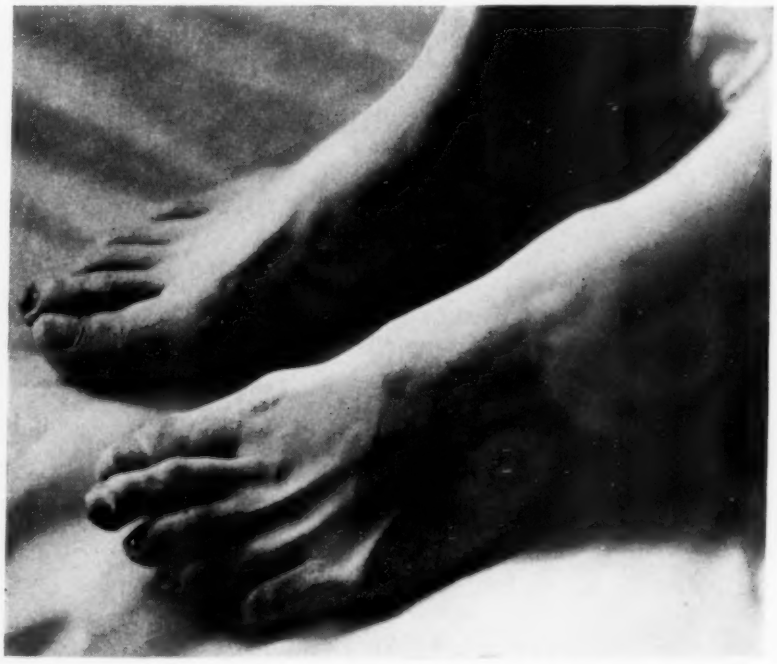

FIG. 2. Gangrene of tip of second left toe at the time of discharge from hospital. Two months before, all toes of both feet were black and appeared gangrenous.

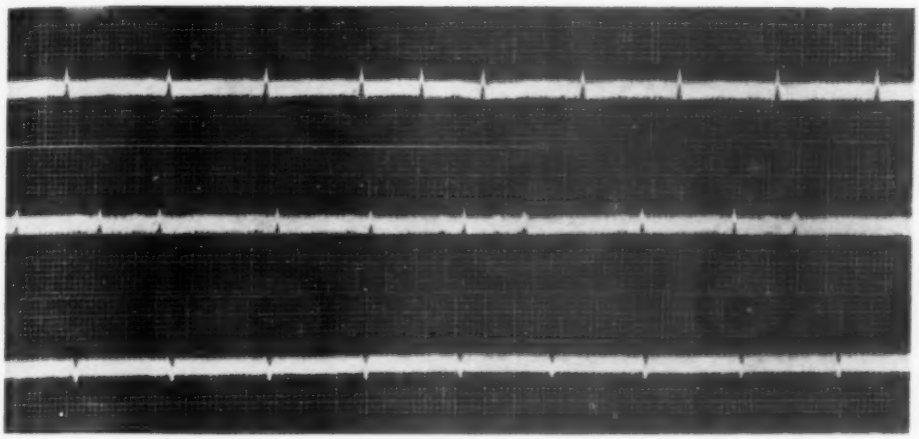

FIG. 3. Electrocardiogram, Nov. 28, 1930. Normal sinus rhythm. Rate 94. Q R S = 0.06 second. T flat in all leads. Left ventricular predominance. Auricular premature beats in leads I and II. Low amplitude (maximum 2.5 mm.).

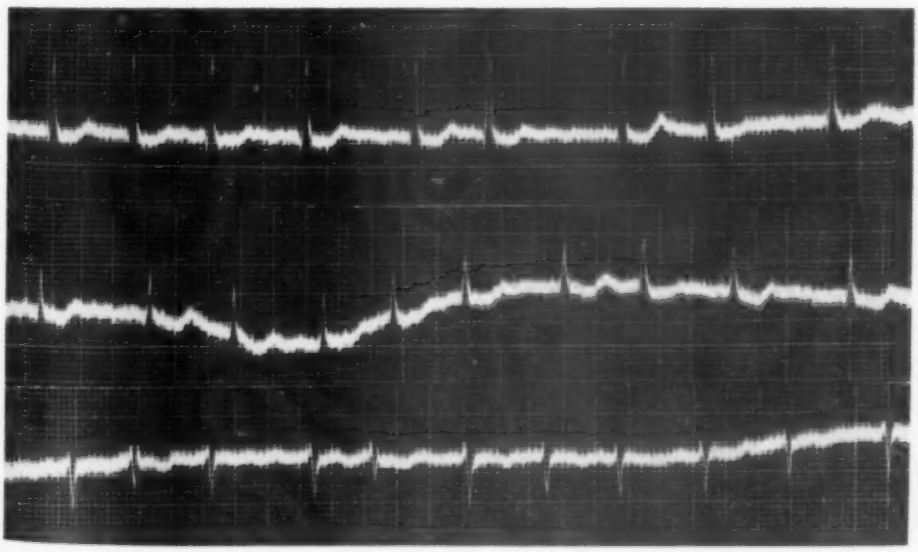

FIG. 4. Electrocardiogram, Dec. 30, 1930. Auricular fibrillation—rate approximately 115. Q R S = 0.08 second. T diphasic in all leads. Left ventricular predominance.

In brief, the sequence of events was as follows: First admitted to hospital in October, 1929, and discharged with the diagnosis of chronic nephritis without edema, and chronic myocarditis, the patient, a woman of 54, was readmitted Nov. 10, 1930, with lobar pneumonia, auricular fibrillation, and chronic nephritis. That evening she received 100 cc. of 50 per cent glucose intravenously, followed by a severe chill and a tremendous fall in the white blood count. On the morning of Nov. 11th her systolic blood pressure was 88 mm., the diastolic 70 mm., and she received one ampule of pituitrin subcutaneously, repeated four hours later. In the next 28 hours she received 24 grains of digitalis leaf, this drug being then discontinued for six days and resumed on the 18th for four days in a dosage of three grains a day. There was a central pneumonia of the lower lobe of the right lung, confirmed by the x-ray. Following the chill on the 11th, the temperature remained normal for three days, and on the morning of the 14th, the blood pressure was 128/88, but that evening the temperature rose to 100° and she received another intravenous injection of 100 cc. of 50 per cent glucose, again with a severe chill, cyanosis, dyspnea, and pains in the calves of the legs. There was again a great fall in the white blood count, from 20,100 to 11,250 in 1½ hours, with return to 25,000, 45 minutes later. 10 minims of 1-1000 adrenalin was injected subcutaneously and beginning then, on the 14th, four or five subcutaneous injections, each of one ampule of pituitrin, were given daily for ten days. On the 17th there was a moderate jaundice, with slight tenderness over the liver, all disappearing after a few days. According to the record, the pulse was totally irregular on all examinations up to Nov 18th and on the 20th the pulse is noted as slow but totally irregular. On the 20th there was observed what was then thought to be subcutaneous hemorrhage almost completely covering the nose, and later that day there occurred burning and pain in all the toes of both feet, the toes being cold, cyanotic and very tender. The next day petechiae appeared on the right hand and what we believed were infarcts of lung. The next record, on Nov. 23rd, records the heart action as regular and slow,

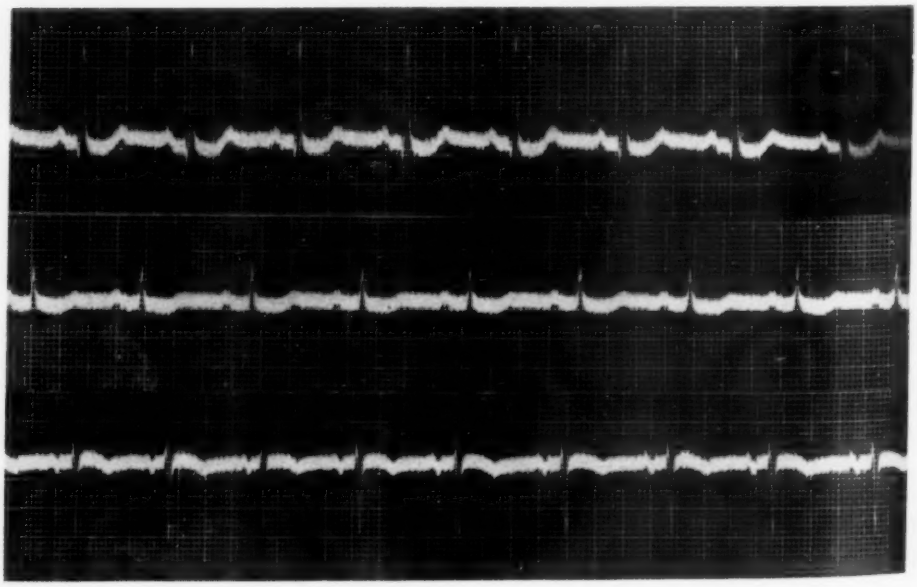

FIG. 5. Electrocardiogram, Jan. 5, 1931. Normal sinus rhythm. Rate 79. P R = 0.18 second. P^2 notched. Q R S = 0.08 second. T^1 and T^2 diphasic, T^3 inverted. Left ventricular predominance.

with beginning gangrene of all the digits of right hand, and "splinter hemorrhages" under all the finger nails of left hand. On the 25th there were discolorations of both feet and it is noted that the dorsalis pedis pulsation was felt on the right foot but not on the left, with no impulse at external or internal malleoli of either foot and no popliteal pulsation in either leg. At this time there was a purple area on the right forearm. One day later, two pustules appeared on the right cheek and these developed in 24 hours into an abscess of the upper lip and right side of the face, which was opened. By this time the lungs were clear. All toes of both feet were black all over and apparently gangrenous. By Dec. 1st, there was a sharp line of demarcation across metacarpophalangeal joints of all fingers of the right hand.

On Nov. 28th, the first electrocardiogram showed a normal sinus rhythm, with auricular premature beats. On Dec. 21st, the patient started again to fibrillate. Digitalis, which had been stopped for about a month, was resumed and the heart action became regular for a few days. An electrocardiogram on the 30th showed fibrillation, as also on Jan. 3rd, 1931, and although on Jan. 5th there was normal sinus rhythm, this did not continue. Accordingly, after careful trial, the patient was put on quinidine on Jan. 7th, and all electrocardiograms taken later, on Jan. 15th and Feb. 3rd, showed normal rhythm. There was no clinical evidence of fibrillation after January 8th.

At the time of leaving the hospital, the condition of the nose and cheek had completely cleared up, all of the toes had healed with the exception of the terminal joint of the second left toe, while circulation had returned in a considerable part of the fingers and thumb of the right hand.

When discharged on March 1st, 1931, the patient felt better than she had for three years or more.

Summary

1. The case is reported of a patient in whom multiple arterial occlusions occurred coincident with the disappearance of auricular fibrillation.

2. It is believed that the return to normal auricular rhythm was related to the use of pituitrin.

3. It is believed that pituitrin should be used cautiously in the treatment of patients with auricular fibrillation.

REFERENCES

[1]Mackenzie, Sir James: Diseases of the heart, 1914, H. Frowde, London, 379, 235.

[2]Hare, H. A.: A therapeutic referendum, 1928, Prog. Med., iv, 311-417.

[3]Meyers, S. G.: Emptying of the gall bladder with especial reference to the use of pituitrin, Am. Jr. Med. Sci., 1928, clxxv, 405-410.

[4]Schondube, W.: Röntgenologische Beiträge zum Entleerungsmechanismus der Gallenblase, Fortschr. a. d. Geb. d. Röntgenstrahlen, 1927, xxxvi, 603-609.

[5]Dale, H. H.: The action of extracts of the pituitary body, Biochem. Jr., 1909, iv, 427-446.

[6]Wiggers, C. J.: The physiology of the pituitary gland and the actions of its extracts, Am. Jr. Med. Sci., 1911, cxli, 502-515.

[7]Ross, J. B.; Dreyer, N. B., and Stehle, R. L.: Cardiac action of pituitary extract, Jr. Pharm. and Exper. Therap., 1930, xxxviii, 451-460.

[8]Kolls, A. C., and Geiling, E. M. K.: Pharmacology of extracts of posterior lobe of pituitary gland, Jr. Pharm. and Exper. Therap., 1924, xxiv, 67-81.

[9]Geiling, E. M. K., and Resnik, W. H.: Action of pituitary tartrate on heart of unanesthetized dog, Jr. Clin. Invest. 1925, i, 239-245.

[10]Halsey, J. T.: Organotherapy and hormotherapy, Endocrinology and Metabolism (Barker, L. F.), 1922, i, Sec. i, D. Appleton and Company, New York, 114-115.

[11]Geiling, E. M. K., and Resnik, W. H.: Action of pituitary extract on the heart of unanesthetized dog, Jr. Clin. Invest., 1925, i, 217-238.

Hereditary Deforming Chondrodysplasia*

By M. A. Spangelberger, M.D., F.A.C.P., *Denver, Colo.*

Hereditary deforming chondrodysplasia is a comparatively uncommon disease of the skeletal system, characterized by bony deformities caused by multiple cartilaginous exostoses and enchondromata (bone cysts). Ehrenfried[1], in 1915, collected over 600 cases from the entire medical literature of the world; Stocks[2], in 1925, brought this number up to 765. Ray[3], in 1930, found 156 cases reported in the American literature and added one of his own. Since then Hyndman[4] has published an interesting case report. The most recently reported case is one by Blount[5] in which a mild rachitis was coincident. This case, with our own, brings the total number of American cases to 160.

Heredity was noted in 83 per cent of the cases reviewed by Ehrenfried, in 75 per cent by Ray and in similar percentages by other authors; however, there remains the minority percentage in which a history of heredity cannot be demonstrated. The fundamental etiological factors are still undetermined. No satisfactory treatment has been discovered.

The exact status of this disorder can be determined only by a larger series of authentic cases, and the following case is herewith reported.

*Submitted for publication, July 13, 1931.

History of Patient

Walter W., age 11, of German descent and a native of Missouri, was the first born of three children. For six months he was breast fed, then the mother, becoming aware of existing pregnancy, weaned him. Several cow's milk formulae were tried. Thereafter a diet of strained vegetables, grain gruels, and the lighter cereals was given. The child began to creep at nine months, and walked before the end of the year. Development proceeded without untoward incident until three and one-half years of age. At this time knoblike excrescences were noted in close relation to the joints of the extremities. These appeared simultaneously, showing most prominently about the knee joints. Locomotion became difficult, and normal movements of the wrist and elbow became limited.

A pediatrist was consulted who made a diagnosis of rickets and prescribed a diet rich in calcium. Cod liver oil was also given, but no improvement followed. Since walking was impossible, a brace was applied, consisting of a belt with metal braces extending along each side of the legs, fastened to soles of specially made shoes. This, too, was unsatisfactory and the child continued resting part of the time in bed and part of the time making his way around with unsteady gait and crawling when balance was lost. At the age of five, improvement ensued. The ability to walk returned, weight increased, and the child entered school at six. Since that time he has been active, taking part in play as a normal child. While he has sustained many falls, there has never been a fracture.

The boy came under observation again, not for the disorder herein described, but as the result of a survey of all grade

school children for faulty breathing. His parents were advised that he required a tonsillectomy. The only other illnesses were pertussis, measles, and mumps at the respective ages of four, six, and eight years. Recovery in each was complete and uneventful.

At the time of the tonsillectomy, a bony spur from the outer condyle of the femur was excised, and a cyst on the anterior aspect of the tibia was curetted.

Father's History. The father was 37 years old, white, of German descent, a native of Missouri, and the eldest of nine children. He was reared on a farm and followed agricultural pursuits until 29 years of age. His height was 6 ft. 4 in., weight 224 lbs., of powerful physique. He had never had severe illness or injury. Minute examination of the skeletal system showed not the slightest disorder. The usual laboratory tests, including Wassermann, were negative.

Mother's History. The mother was the second of five children, age 35 years, a native of Missouri, and of German descent. She married at 19 and gave birth to this child one year later. Two other children were born 11 months and three years later. She was reared in the country and had the usual childhood diseases with uneventful recoveries. Her health was excellent until five years preceding examination when pulmonary tuberculosis developed. A change of residence was advised, and the family then came to Colorado. Her disease became quiescent and was arrested at the end of two years. At that time she had regained her normal weight of 130 pounds and was apparently in a fair state of health, when a Graves' syndrome began with a rather sudden onset. Thyroidectomy was done and after several months apparent recovery was established. Physical examination now shows a cavitation in the upper right lung, but no signs of activity. Basal metabolic rate is minus two. Usual laboratory tests including Wassermann, negative. As in the instance of the father, a careful examination of the osseous system disclosed no abnormality.

Correspondence with relatives of both parents fails to elicit evidence of any gross abnormality such as exostoses. Nor is there any report as to unusually short stature or limitation of joint movements.

Physical Examination of Child. The patient is a fairly well nourished boy; height 48 inches; weight, 68 pounds. As he stands nude (figures 1 and 2) misshapen joints are strikingly apparent. Knoblike protuberances, of varying size occur at the elbows, wrists, knees, ankles and about the chest and pelvis. These are larger about the joints of the left side of the body. Manu valgum and genu valgum exist, and these, too, are more marked on the left side. There is a varying amount of limitation of movements in all major joints; he is unable to flex the left thigh on body; pronation and supination of the left hand cannot be fully accomplished. The gait is awkward, but the patient is remarkably agile, despite the disadvantage of distorted joints.

Head. The head is symmetrical, the face proportionate, and no exostoses are evident. The hair is fine and abundant, with clean scalp. The skin is warm, smooth, of excellent texture and color.

Eyes. Blue, pupils round, equal, react to light and accommodation. A diopter of myopia is measured in each eye. There exists no muscular imbalance. The fundi are normal.

Ears. Normal.

Teeth. Of good structure, well spaced, and dentition has progressed normally for age.

Throat. The tonsils are hypertrophic, exhibiting crypts containing caseous material. A pad of adenoid tissue is visible in the posterior-pharynx.

Thorax. The thorax is flattened, but respiration is adequate and equal. There are no adventitious sounds on auscultation of the lungs.

Heart. Normal outline of the heart is found on percussion. The apex is in the fifth interspace, one-half inch within the nipple line. The heart tones are normal. There is a brief presystolic murmur at the apex.

Abdomen. The abdomen is flat, soft, and no organs or masses are palpated.

Nervous system. All superficial and deep reflexes react normally.

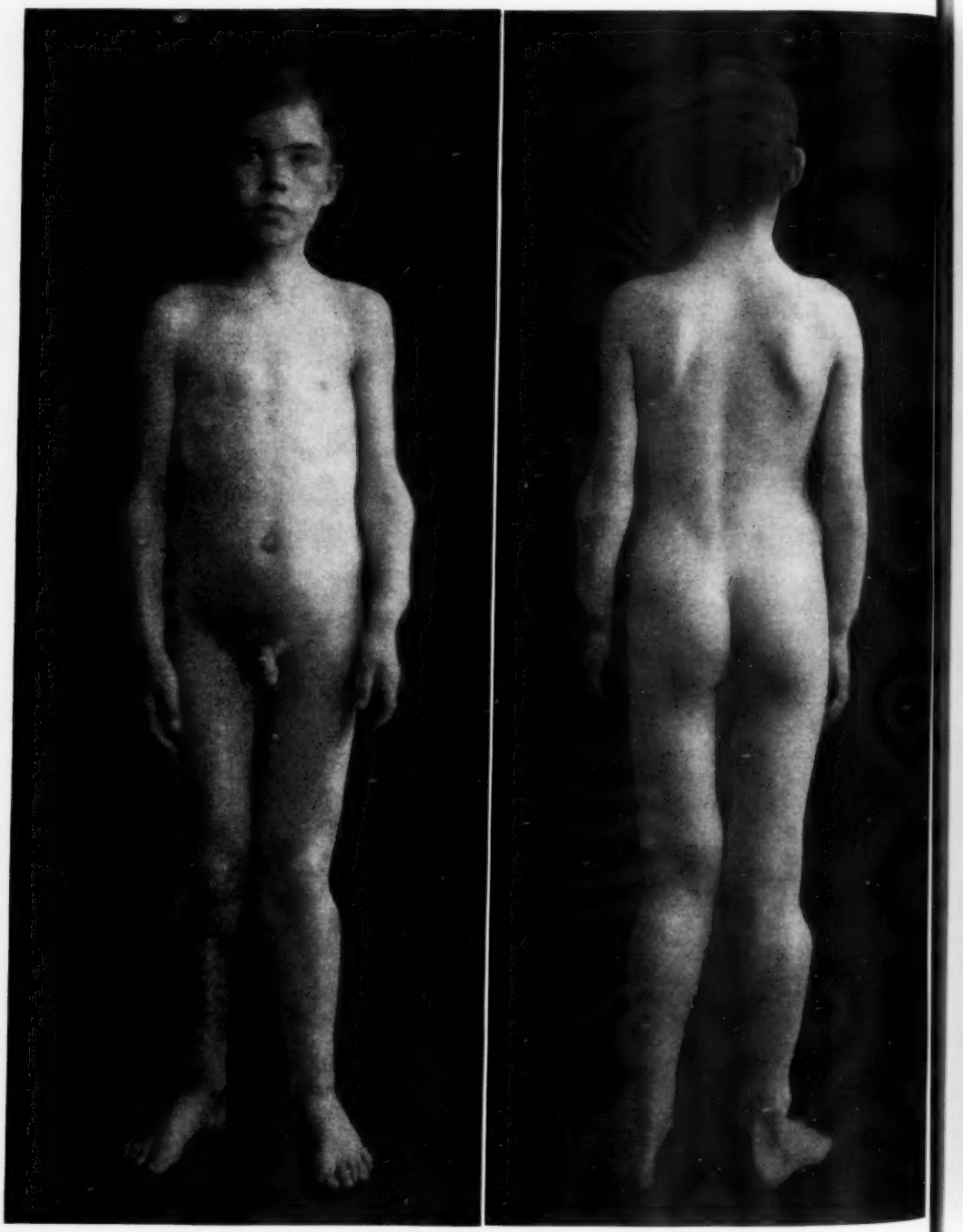

FIG. 1 FIG. 2

FIGS. 1 and 2. Anterior and posterior views of patient to show skeletal deformities.

Skeletal System. The findings are based upon physical examination and a complete x-ray examination of the entire skeleton.

Skull. There are no exostoses, enchondromata or deformities in the bones of the skull and face.

Spine. The spine is straight. Nearly all the transverse processes are involved.

Ribs. There is an exostosis on the left fifth rib, near the spine, with some pressure deformity of the fourth rib above this exostosis. A larger growth can be felt and seen on the seventh rib in the right midaxillary line. There are also several smaller deformities on the anterior ends of several ribs.

Pectoral Girdle. There are several exostoses along the superior margin of the scapulae and the glenoid fossa is involved. The acromial end of each clavicle is broadened and irregular.

Pelvic Girdle. There are several exostoses on each ilium. The acetabulum on both sides is broadened and shallow. The texture of the pelvic bones is coarser than normal.

Upper Extremities. The humerus on both sides is broadened at the upper end and the shaft of the bone is bowed outward. The bones of the forearm on both sides are shortened and bowed, the ends broadened and distorted and there are large exostoses at the lower ends. These deformities are most marked on the left side and have resulted in dislocation of the upper end of the radius. Carpals, metacarpals and phalanges show abnormal variations in shape and size. Several exostoses and enchondromata are noted. (Figure 3.)

Lower Extremities. The neck of each femur is markedly straightened and broadened. The trochanters are widened and thickened. The texture of the upper end of the shaft is abnormally coarse. The lower ends are broad and thick, the contour is irregular and there are several clubbed stalactite growths pointing away from the epiphyses. The upper end of each tibia shows the same characteristic stalactite exostoses as described for the femur. The upper end of the fibula is broadened and thickened, almost twice normal size, irregular in contour and a large irregular osteoid formation is noted on the posterior surface. (Figure 4.) The lower end of the diaphysis of the left fibula is expanded to three times normal diameter by a large enchondroma; a similar deformity, not quite so marked, is noted on the lower end of the right tibia. The bones of the feet show about the same degree of pathological change as the bones of the hands.

Laboratory Examinations. Hemoglobin, Dare, 92 per cent; red cells, 4,100,000; white cells, 5600; Wassermann test negative. Several estimations of blood calcium gave values within normal limits. Urine: acid; sp. gr., 1.020; sugar, bile, acetone, and indican tests were negative. No pus cells, red cells or casts were seen; and crystalline deposits were normal. The test for Bence-Jones' protein was negative.

Discussion

Since the various features of this disorder have been thoroughly discussed in many papers we shall consider only those which have a direct bearing on this case.

The physical findings establish the diagnosis. The short stature, the shortening of the extremities with deformities at the elbows, knees, wrists, and ankles, the inability to extend the forearms fully, the multiple palpable and, in part, visible exostoses near the ends of the long bones are characteristic. The roentgengrams show many enchondromata and many spurs, arising chiefly from the metaphyses near the epiphyseal junctions. The x-ray pictures of the enchondromata have the soap bubble appearance of a benign giant cell tumor. In giant cell tumor exostoses do not develop, the disease is progressive, accompanied by much pain, and eventually results in fracture, extension by spontaneous perforation or surgical interference. Simple, non-hereditary

osteomata, chondromata and enchondromata are usually single. According to Honeij[6] the bones in chondrodysplasia are very sensitive to trauma and easily respond by formation of exostoses. Osteitis fibrosis cystica does not produce the exostoses and the curettings of a cyst determine the diagnosis.

The case under discussion was diagnosed by several consultants as rickets. This diagnosis seems plausible in view of the history of feeding by the mother until she was five months pregnant. Roentgenograms at this time might possibly have determined the correct diagnosis.

Hyndman[4] gives a very satisfactory differential diagnosis between chondrodysplasia and chondrodystrophia fetalis. He rightly points out that the latter is a generalized disease usually involving all the epiphyses equally and producing a symmetrically dwarfing skeletal deformity which is usually well developed at birth and therefore dates back to intrauterine life.

Although true heredity and familial incidence in this disease has been unquestionably demonstrated by many

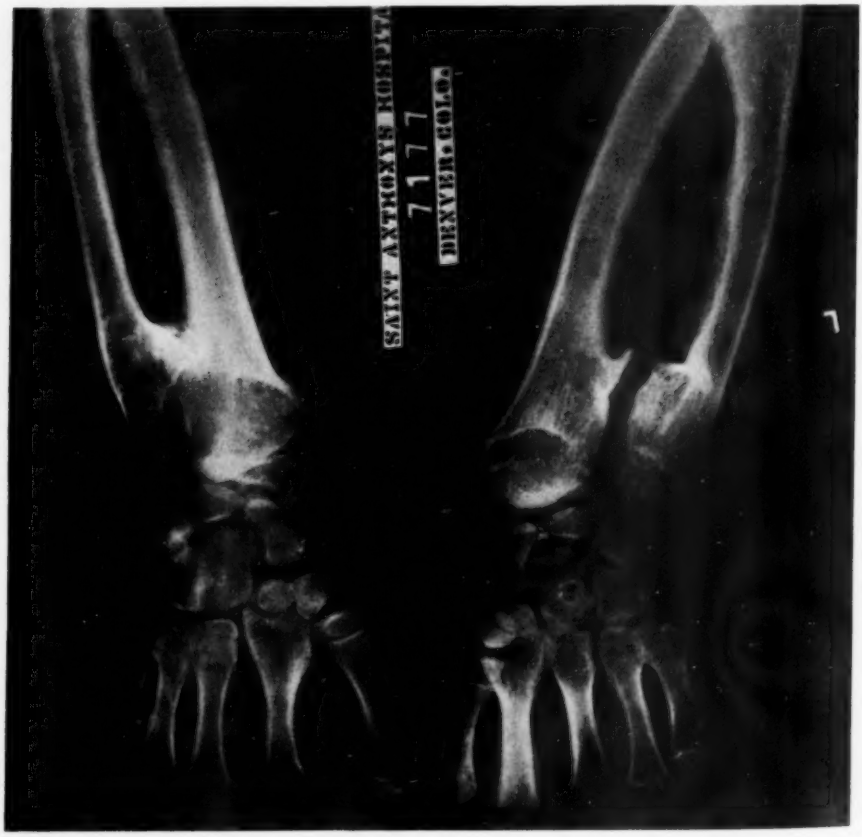

FIG. 3. X-ray of forearms. Note shortening and bowing, and exostoses and enchondromata.

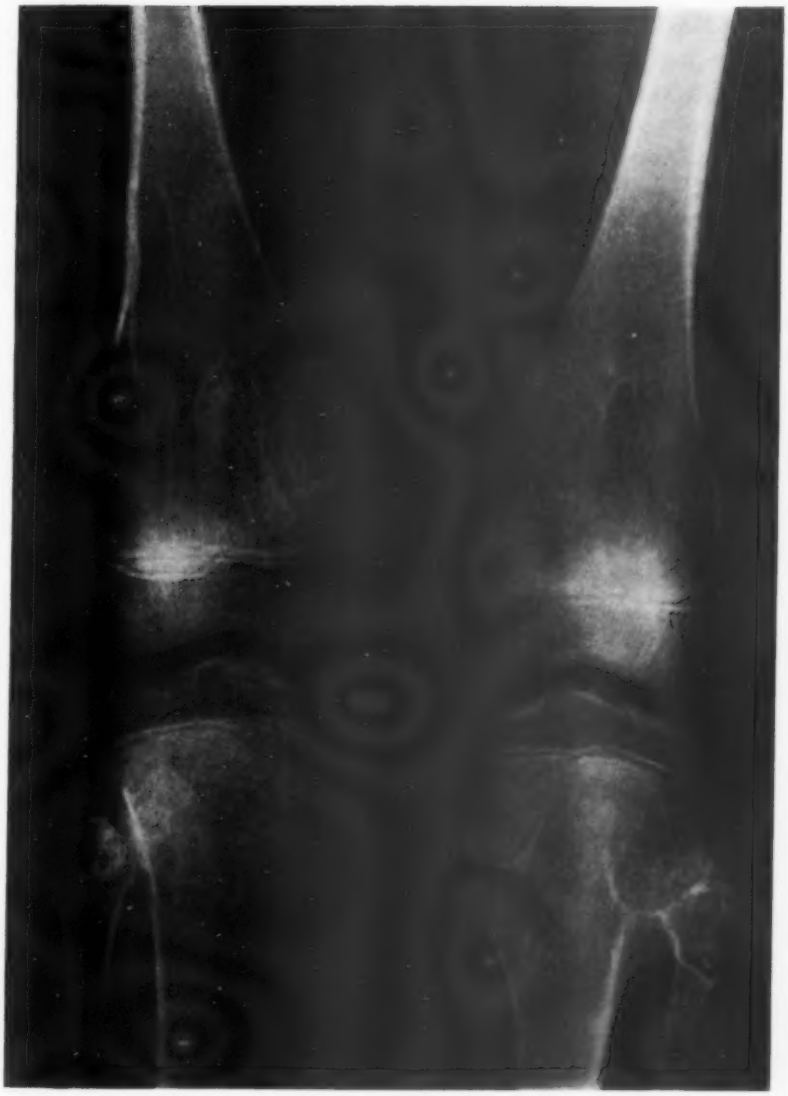

FIG. 4. X-ray of knee joints. Note enchondromata and stalactite exostoses.

authors, a careful search into the family history of the patient revealed no other cases. However, a hereditary history is often lacking, as in this case. Possibly the stigmata of this disease may at times be so insignificant that they would be overlooked except on the most careful physical examination, including a complete x-ray examination of the skeletal system. It may also be that those cases in which actually no hereditary basis exists can not be considered as true examples of this disease, even though the physical findings are typical. Ehrenfried[1] coined the name "hereditary deforming chondrodysplasia"; other authors have used other names because they did not agree with his conception of the major features. It may finally be that the hereditary factor is not found in some disturbance of the bone anlage, but in some disturbance of the endocrine system. In that case the changes in the skeleton may appear only in certain cases of such endocrine disturbance. In our case there is a history of very severe exophthalmic goiter during the pregnancy of the mother. Jegat[7] suggests that tuberculosis may have an etiological significance. Although there is a history in this case of tuberculosis in the mother, the evidence for such a conclusion is unconvincing. The importance of heredity in this disease has been established be-

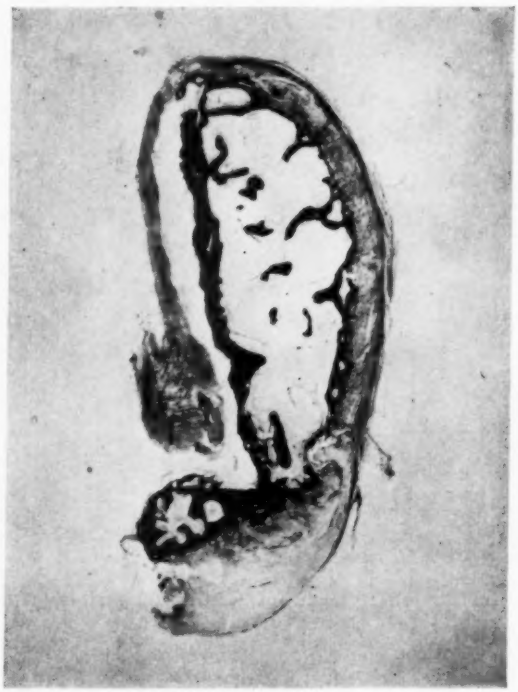

FIG. 5. Cross section of exostosis from left femur. Note disorderly growth of cartilage and bone.

yond all reasonable doubt. But is it not possible that the hereditary factor is to be found in an underlying disturbance of the endocrine system?

Whatever the real cause may be it results in a chaotic growth of the metaphyseal-epiphyseal line. The deformities resulting may in part be due, as Keith[8] has suggested, to a failure of proper pruning of the ends of the long bones and for this reason he calls this disease "diaphysial aclasis".

Microscopically, the picture presented is one of disorderly cartilaginous growth with irregular ossification as shown in figure 5, a cross section of an exostosis, and in figure 6, curettings from an enchondroma in the lower end of the femur. These sections correspond to the pathologic changes so beautifully described by Ehrenfried[1] and Honeij[6].

Symptomatology in this disease depends upon interference by the deformity with motion, blood and nerve supply, upon secondary changes such as inflammation and upon true tumor formation. Hyndman[4] believes that all cases of hereditary deforming chondrodysplasia should be carefully checked from time to time to detect immediately any possible true neoplastic changes. If such occur he advises prompt and complete surgical excision.

Sulphur, phosphorus, calcium, and magnesium determinations in the blood and the urine have so far not given any clew as to rational treatment in this disease. Certainly calcium therapy does not produce the beneficial effects that it does in rickets. If the disease is a true hereditary dsease determined in the bony anlage perhaps no satis-

FIG. 6. Curetting from enchondroma of left tibia. Note atypical cartilage and abnormal bony growth.

factory treatment will ever be discovered.

SUMMARY

A case of hereditary deforming chondrodysplasia is reported.

No evidence of hereditary origin could be discovered.

There was a history of exophthalmic goiter and of tuberculosis in the mother.

REFERENCES

[1]EHRENFRIED, A.: Multiple cartilaginous exostoses — hereditary deforming chondrodysplasia. A brief report on a little known disease, Jr. Am. Med. Assoc., 1915, lxiv, 1642-1646.

EHRENFRIED, A.: Hereditary deforming chondrodysplasia. Multiple cartilaginous exostoses. A review of the American literature and a report of twelve cases, Jr. Am. Med. Assoc., 1917, lxviii, 562-568.

EHRENFRIED, A.: Hereditary deforming chondrodysplasia. More cases. Am. Jr. Orthop. Sug., 1917, xv, 463-478.

[2]STOCKS, P.: Hereditary disorder of bone development. In: The treasury of human inheritance, 1925, 3, Cambridge University Press, New York.

[3]RAY, H. H.: Diaphysial aclasis: hereditary deforming achondrodysplasia. Arch. Ped., 1930, xlvii, 152-164.

[4]HYNDMAN, OLAN R.: Hereditary deforming chondrodysplasia. Report of a case. Arch. Surg., 1930, xxi, 12-31.

[5]BLOUNT, W. P.: Chondrodysplasia. An unusual case. Am. Jr. Dis. Child., 1930, xl, 327-333.

[6]HONEIJ, JAMES A.: A study of multiple cartilaginous exostoses. Four cases with report of calcium and magnesium metabolism in two cases. Arch. Int. Med., 1920, xxv, 584-627.

[7]LENOBLE, M. E., and JEGAT, M. Y.: Trois cas d' exostoses ostéogéniques héréditaires et familiales. Bull. et mém. Soc. méd. d. hôp. de Par., 1922, xlvi, 1661-1669.

[8]KEITH, A.: The true nature of the condition known as multiple exostoses, Tr. Med. Soc. Lond., 1920, xlii, 67-74.

The Dietetic Control of Some Forms of Hypertension and the Associated Gastrointestinal and Nervous Symptoms*

By V. C. Rowland, M.D., F.A.C.P., *Cleveland, Ohio*

IT IS extraordinary, in view of the available facts, that the profession has so largely ignored the therapeutic possibilities of diet and weight control in certain forms of hypertension associated with plethora and gastrointestinal and nervous symptoms.

By way of introduction, a brief reference to the literature of hypertension will be of interest.

From the time of George Johnson in 1868, and of Gull and Sutton in 1872, the subject of vascular disease has been continuously studied, and approached mainly from the pathological viewpoint. After much controversy as to the characteristic changes in the intima and media, there seems to be fairly general agreement with the views of Fischer and Schlayer in 1910, and of Brogsitter in 1924, and of Kernohan, Murphy and Grill in 1930, namely, that the primary change is an hypermyotrophy of the media or at least a thickening of the arterial muscle wall with an increased number of nuclei which may be interpreted as a hypertonic contraction in response to some hypothetic pressor substance, or some stimulus to the sympathetic nerves. The thickening of the muscular layer of the vessels is not necessarily associated with any primary degenerative change. Later there may be proliferation of the intima and subsequent hyaline or other degeneration. Kernohan, Anderson, and Keith, in 1930, reported an extensive study of hypertension from biopsies from muscular tissue showing a similar thickening of the muscular layer. Ophthalmoscopic evidence, such as that of Volhard in 1931, also seems to confirm the idea of a primary contraction and only subsequent degenerative change.

There have also been a few disconcerting cases of essential hypertension with death by cerebral or other accident, with absolutely negative pathological findings. These cases, together with much clinical evidence, suggest the conception of Clifford Allbutt, that there is a functional stage of hypertension, and that the characteristic pathology of diffuse vascular disease is the result rather than the cause, contrary to current pathological opinion. Furthermore, it is generally agreed that there is no sharp classification of the different forms of hypertension, that they differ mainly in degree, that

*Given before the American Gastro-Enterological Association at Atlantic City, May 4, 1931.

either benign or malignant hypertension may run a progressive course with very moderate elevation of blood pressure, and that much advanced vascular sclerosis may have no elevation of blood pressure whatever.

The literature of obesity also is important as a background to the following report upon the dietetic management of some forms of hypertension. Insurance statistics, largely ignored by the medical profession, have for years repeatedly confirmed the association of increased blood pressure and high mortality rates with obesity. The Actuarial Society of America, in 1925, published a compilation of the results of the statistics of twenty-six companies, and a similar forthcoming compilation of a larger and better controlled set of statistics, it is said, will confirm, that approximately two-thirds of all individuals over forty years of age, who are ten pounds or more overweight, show a definite elevation of blood pressure. Similar figures were shown in private studies of obesity, such as that of a thousand cases in Boston by Preble in 1923. There is some statistical evidence, also largely ignored, of the lowering of blood pressure by reduction in weight.

Furthermore, the literature of the more scientific type on the subject tends to the idea that obesity is largely exogenous (Newburgh). Students agree, as Bauman states, that there is no inherent change in the basal metabolic rate, except in the subthyroid group. The work of Grafe and others points to a compensatory rise in the basal metabolism during periods of overfeeding. Increased metabolism implies increased blood pressure and pulse rate. There is, of course, a type of obesity with low blood pressure and low metabolism. However, as Evans has recently shown, if basal metabolism rates are calculated on normal rates for the height and age of obese individuals, instead of actual weights, many of these rates will be high instead of low, and thyroid medication clinically unsatisfactory, as might be expected. There is evidence pointing to a lower specific dynamic action to protein in certain obese individuals. This is not constant, however, and can only be one factor in the obesity, possibly due to deficiency of anterior pituitary function (Liebesing).

The type of patient who presents the form of blood pressure most amenable to dietetic control is one in his forties or early fifties, definitely overweight, ruddy, strong, and active, eating heartily of rich food, not too fond of muscular exercise, and not too highly nervous. He is usually seen in private practice in the so-called higher social levels, although in our country and generation, even in hard times, high caloric food, if not wholesome balanced diet, is freely available and obesity abounds well down the social scale. The patient is either symptom free or beginning to show minor nervous and digestive symptoms such as palpitation, headache, heartburn, and flatulent indigestion. Frequently he has gained twenty or thirty or more pounds over his optimum weight at twenty-five or thirty years of age. He may complain of lack of initiative or slight lack of endurance in comparison with his earlier working capacity. If his blood pressure had been observed, there was considerable fluctuation with

a general tendency toward a higher and more continuous blood pressure level. Frequently, but not invariably, there is a family history of the apoplectic habitus and high blood pressure or of obesity and diabetes. The most suitable cases show no organic pathology or very early signs of cardiac enlargement and aortic fullness fluoroscopically. Presumably, the hypertension is due to a functional arterial hypertonus or that with an early and often controllable stage of diffuse vascular disease. Our ideal patient is intermediate between the young individual with very high systolic and diastolic pressure and the older patient with advanced arteriosclerosis or marked kidney pathology.

As indicated below, a reducing regimen in this group of cases effects a very prompt reduction in blood pressure and by maintaining weight at or near a normal level, permits of sustained control of the blood pressure to a degree not generally appreciated. There is, apparently, also a functional moiety in the hypertension associated with advanced cardiovascular disease which may be gradually reduced to the optimum level as determined by the increased peripheral resistance. For purposes of practical therapy, therefore, the most useful classification, if not the clearest in the present confusion of the subject, is that of functional hypertonus and fixed hypertension of organic peripheral obstruction.

With this introduction, we may approach the following observations upon the relationship between diet, weight, hypertension, and associated symptoms.

From personally kept records of private patients over a period of years, one hundred cases were selected for this study. All blood pressure observations were made personally by the writer under the same conditions. In the tabulation, part of which appears below, the first weight given is the weight of the patient at the beginning of management; the second is the patient's statement of weight in early adult life in health, usually about twenty-five years of age, as a guide to normal weight; the third is the weight at the end of a period of management, mainly by diet. This was a balanced diet of milk, egg, meat, vegetable, and fruit, simply low in sweets, fats, pure starches and total calories. Huchard's idea that the quality of food stuffs is more important than the quantity has never been supported. However, excess of protein was avoided in our diets. Salt restriction played no part, although seasoning, condiments and stimulants were interdicted during the period of management. Frequently a prompt drop of five or ten pounds is noted the first week. Later, however, one pound a week was the goal. In all cases, reduction was kept at a rate which gave an improved sense of well being, not weakness or distress. The beginner was often hungry with the sudden change in his habits of eating. In fact gastric analysis in a few cases showed hypersecretion and hyperacidity in comparison with readings taken after there had been time for adjustment to the reduced diet. Clinically too, after this adjustment, many patients testified that they no longer had the craving for excessive food intake. A minimum of medication was used, usually a little bromide or luminal for

short periods for symptomatic relief. Thyroid extract likewise was used only occasionally and in small dosage. No nitrites, sulphocyanates, calcium, atropine, bismuth salts, or other drugs were used in the reported cases. None of the cases recorded were taken off their usual schedule of work. Associated conditions were noted such as indigestion, ulcer, gall bladder disease, diabetes, thyroid disorders, menopause, cardiovasculorenal disease, nervous disturbances, urticaria and other eruptions, focal infection, arthritis, etc.

A sample of the method of tabulation of data follows together with individual and composite graphs of weight, systolic and diastolic pressure, and, in diabetes, of blood sugar.

TABLE I.

No.	Sex	Age	Weight	Blood Pressure	Associated Disorders	Urine	Blood Sugar	Period of Treatment
9	F	70	190	210/110	old hemiplegia	neg.	140	2/27/30
			150		arthritis			
			161	130/80				4/24/31
19	F	66	172	190/95	much pyrosis	alb.	180	2/10/23
			120			ft.		
			135	130/80			109	3/19/28
25	F	70	151	230/90	sour eructations		153	8/ 7/29
			95		nausea			
			135	140/70			95	1/23/31
28	F	51	200	200/95	exhaustion	sugar	240	10/12/28
			125		edema of feet	cystitis		
			166	130/80				11/13/30
34	F	60	179	215/110	vertigo	casts	88	10/26/28
			135		familial	alb.		
			151	140/80	hypertension			12/15/28
41	F	58	138	170/90	neuralgias	sugar	108	7/10/30
			115			once		
			123	120/75				4/20/31
58	F	49	160	215/110	pyrosis	neg.		2/ 8/30
			125		dent. inf.			
			140	115/80	palpitation			8/ 8/30
56	M	62	190	220/140	cardiac	alb.		1/24/27
			150		enlargement	casts		
			158	160/90	gall stones			12/31/30
60	F	68	180	170/85	diabetes		106	2/ 3/31
			130					
			151	140/68				3/20/31
64	F	50	151	150/90	indigestion	alb.	101	10/12/29
			123		urticaria	ft.		
			127	130/80				2/28/30
67	F	68	143	250/90	indigestion	neg.		2/11/29
			98		flatulence			
			124	170/75				7/15/29
68	F	50	144	170/100	goitre	neg.	100	12/ 4/28
			120		Wassermann++++			
			132	130/87				3/12/29
73	M	42	202	160/80	chronic	neg.		12/17/30
			185		indigestion			
			177	120/80				1/28/31
79	F	50	190	210/100	indigestion			3/24/24
			...		"rheumatism"			
			157	120/80				4/24/31
100	F	59	168	220/110	hereditary			4/13/27
			125		hypertension.			
			164	135/78	angina pectoris			4/25/31

From the family histories, it is very apparent that heredity plays a definite rôle in the individual tendency to hypertension and also to obesity, but instead of accepting this as inexorable, efforts at prevention are shown to be very definitely worth while. As in diabetes, also with an hereditary predisposition, the latent tendency need not always become manifest. On the contrary, when carbohydrate metabolism is impaired full control allows improved function. Ten cases of diabetes are listed among the hyperten-

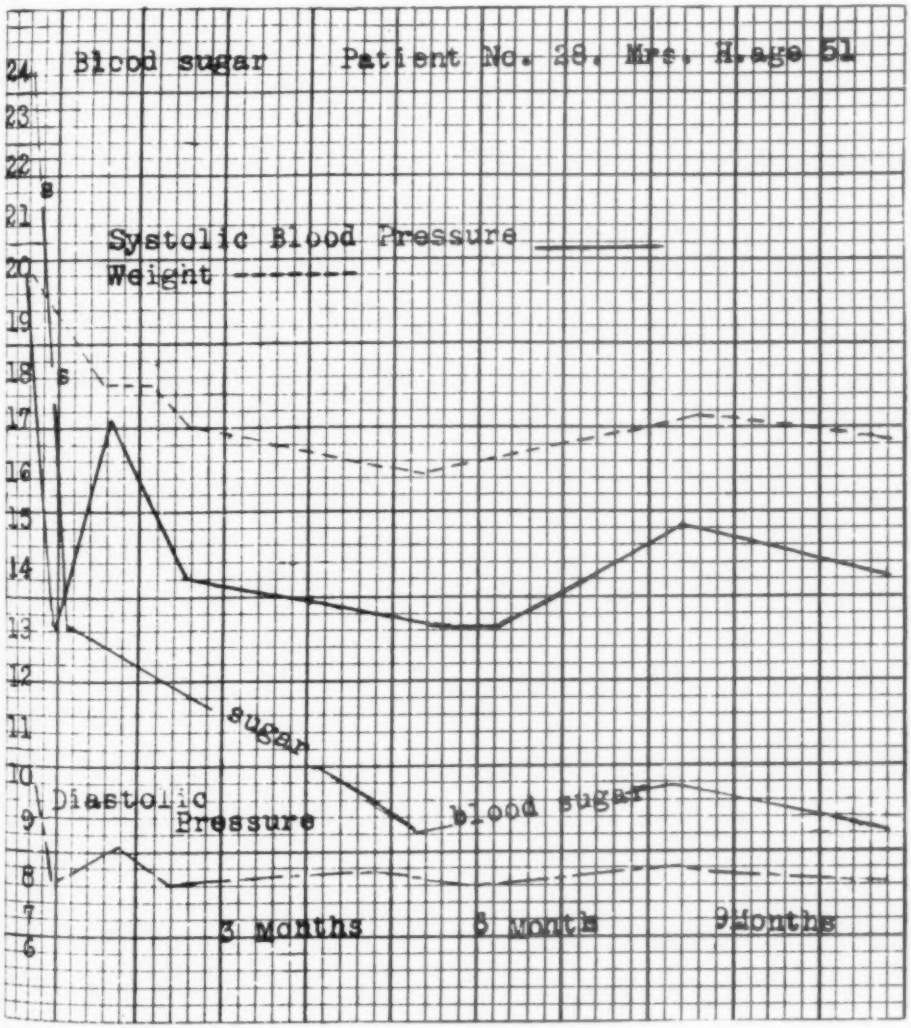

CHART 1, Patient 28. A woman of 51 years with an initial weight of 200 lbs., blood pressure 200/98, blood sugar reading of 240 mg., and sugar in the urine. Under dietetic management and observation for one year, final readings were: weight, 166; blood pressure, 138/80; blood sugar, 90. Relief from weakness, dyspnea and palpitation.

sion cases and the hypertension curve nearly parallels the hyperglycemic curve.

Typical cases of weight reduction with prompt response in blood pressure are shown graphically. The range in total weight reduction was 0 pounds to 54 pounds and in systolic blood pressure from 10 mm. of mercury to 105 mm. The average weight loss for the 100 cases was 20.06 pounds. The average drop in systolic pressure was 40.85 mm., in diastolic pressure, 17.32 mm. The average period of management was 8.28 months. Many of these cases were seen at long intervals for years afterwards with blood pressures controlled in proportion to their weight control. Preliminary to this report (April, 1930) a group was rechecked. The following is an example:

Case 10, a woman seen in 1924, with a blood pressure of 210/110 and weight of 190, now shows a pressure of 120/80 and weight of 157, at 57 years of age. She has entirely and very intelligently changed her habits of eating, has lost all craving for rich desserts and heavy eating, but has continued at her same occupation during the whole period of seven years.

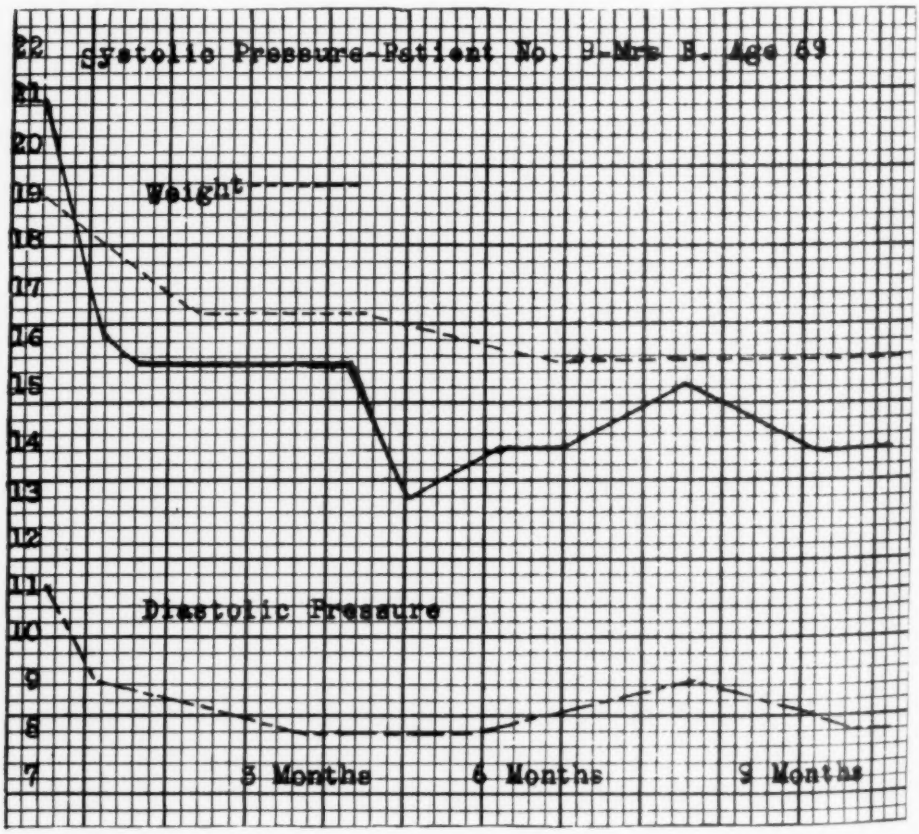

CHART 2, Patient 9. A woman of 69 with initial weight, 190; blood pressure, 210/110. First year of management reduced weight to 155 and blood pressure to 140/82. Relief from severe neuralgias and "rheumatism."

It is recognized that some error occurs in certain cases in that the first readings may be high on account of nervousness and acute upsets which suggest their seeking medical advice at the time. At best there are fluctuations which must be averaged over a period of time. With care, however, this error can be fairly well discounted and with observation over a long period, the reaction of a given patient to strain, to the menopause, and to acute infections can be noted and the blood pressure compared with the original reading. In the cases reported, no factor was as important as that of weight. After full adjustment at the reduced weight levels, the blood pressure was much more constant even under extra strain.

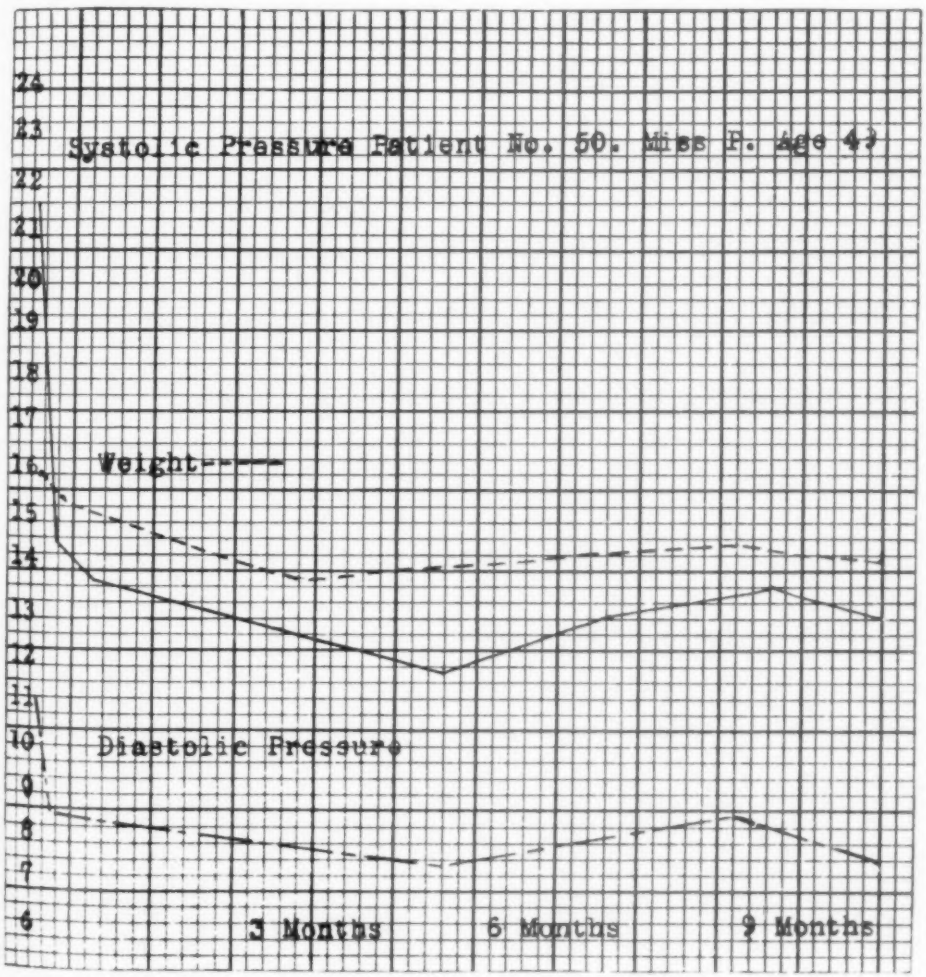

CHART 3, Patient 50. A woman of 49 with initial weight 160; blood pressure, 215/110. First year of management reduced weight to 140 and blood pressure to 130/78. Relief from weakness and indigestion.

Case 41, for example, is a teacher of 58, seen first in vacation time, July, 1930, symptom free at the time although she had had much neuralgic pain. She did not appear to be much overweight at 138 pounds but she was eating heartily and her early adult weight was 115 pounds. Under management she very gradually reduced to 123 with blood pressure reduction of 170/90 to 125/75 which remained quite constant during the following teaching year, the last observation being on April 18, 1931.

Relapses with marked increase in weight as in case 56 (shown in the graphic record) show the direct effect of increased weight on blood pressure after a prolonged period of control. Relapses are not so common as might be expected, especially among intelligent people under intelligent and conscientious supervision and with proper regard for normal balance and variety in diet.

Associated Symptoms

Nervous and digestive symptoms most commonly accompany the hypertension. Of the former, headache, neuralgias, paresthesias, vasomotor instability, palpitation, insomnia, nervous irritability to the point of a definite psychosis, all have occurred and

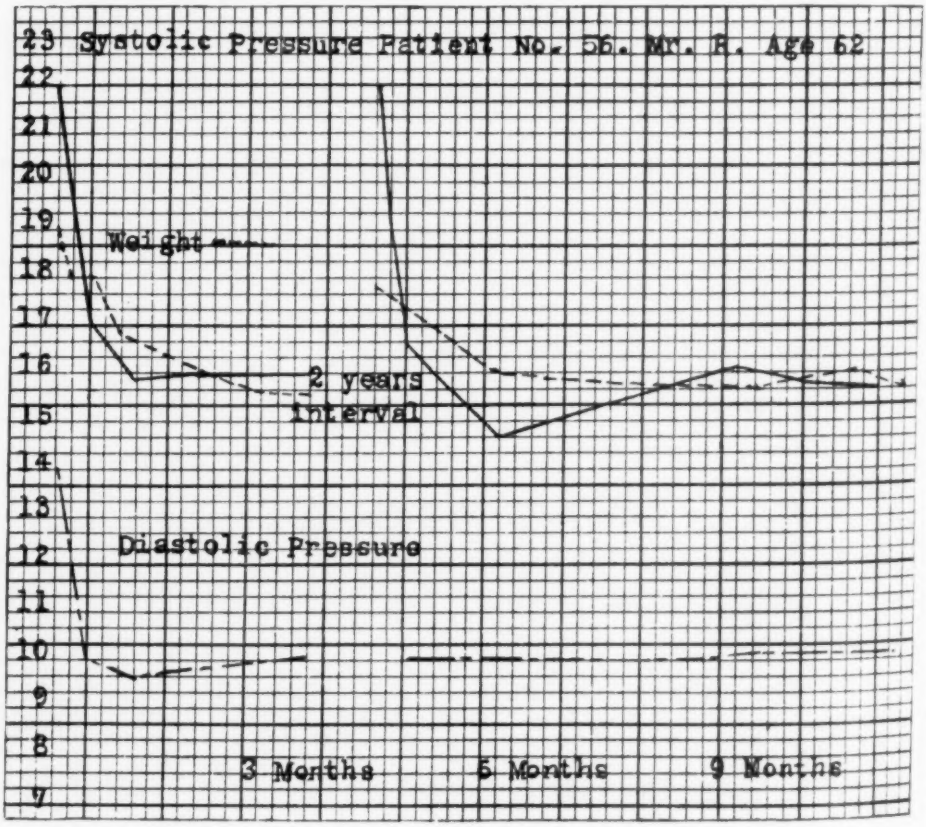

Chart 4. Patient 56. A man of 62, initial weight of 190, blood pressure 220/40. Reduced in 4 months to 155 pounds and blood pressure of 160/100. Relapse for 2 years, gaining weight to 176 with pressure of 220/100. Maintained for 6 months at 160 to 155 pounds with pressure 145 to 160/100.

have been relieved to an extraordinary degree.

Digestive symptoms range from simple distress, pyrosis, eructations, flatulence, nausea and vomiting to a frank gallbladder type of dyspepsia or ulcer syndrome. An analysis of the histories showed seven per cent of clinical diagnoses of gall bladder disease, three confirmed by operation; and four similar diagnoses of ulcer, none of which were confirmed by operation. It is quite probable that more cases had had at some time during their history definite peptic ulcer, since the symptoms were often highly suggestive. X-ray studies were not made in cases that responded promptly to dietetic management of the hypertension. This suggests caution and individualization in the treatment of ulcer so that the older ulcer patient with a tendency to weight and hypertension is not eventually hurt more than helped by frequent high fat feedings such as the full Sippy diet.

Furthermore, there occurs as a complication of hypertensive cardiovascular disease a very intractable form of ulcer with deep craters and rapid erosion leading to hemorrhage or perforation. Autopsies often show these lesions associated with advanced circulatory pathology. Sometimes the ulcer seems to be of the nature of a terminal complication. Accordingly, whatever aggravates the hypertension and circulatory strain, such as a high caloric diet, would be a bad form of ulcer treatment.

Healed ulcer cases who gain unduly in weight often show symptoms suggestive of recurrence. That one can make this diagnosis too hastily is shown by a case in which the writer had an unusual opportunity due to the accidental death of the patient of showing a scaphoid cicatrical fossa in the duodenum with no hint of recent ulceration or inflammation. Deformity of the duodenum had been apparent by x-ray shortly before death, similar to that observed several years before following treatment for typical symptoms and profuse hemorrhage. His recent symptoms were apparently due to heavy eating *per se*. The case is instructive as suggesting that the ulcer syndrome even in a former ulcer patient may precede actual ulceration, and that the latter is but an incident in, or a complication of, a primary metabolic, or other disorder.

Gallbladder disease with its relation to the cholesterol metabolism and its frequent association with the overfed type of patient is even more closely related to hypertension. Likewise very suggestive symptoms may result from the metabolic disorder *per se* without organic disease demonstrable even at autopsy. Many of these symptoms are relieved by the reducing management. In the above 100 cases, there were 26 cases with chronic indigestion of varying degree besides the 7 diagnosed as definite gall stones or gallbladder disease. At least 22 of the 26 were satisfactorily relieved.

The gastroenterologist has a great therapeutic opportunity in the simple dietetic management of these cases. The incidence of hypertension and excessive weight is enormous and apparently increasing in our generation. Janeway found 11.1 per cent of 7872 cases in private practice with blood pressures above 165. Deaths from

hypertension and its complications in the United States are said to number 140,000 annually. Diet has gotten into disrepute because of the many weird abuses under that name, but will undoubtedly play a larger rôle in health supervision in the future. With the new emphasis on preventive medicine, now arrived at the stage of individual participation, we may expect the matter of diet to be brought more in line with medical science.

Summary

1. The literature of essential hypertension indicates that the primary structural change is an hypermyotrophy of the media of the arterioles and that degenerative changes are secondary. Functional hypertonus occurs; in rare cases without demonstrable pathology even at autopsy. A hypothetic pressor substance of metabolic or glandular origin is postulated as the causative agent.

2. The literature of obesity shows an unmistakable association with hypertension. Approximately 66 per cent of all people over 40 years of age and 10 pounds or more over weight show hypertension.

Obesity is largely exogenous.

Metabolic rates are usually normal; sometimes increased.

Mortality rates are increased in proportion to the excess in weight.

3. Heredity is a strong predisposing factor in hypertension and obesity as in diabetes and other diseases.

4. A balanced reducing diet intelligently supervised is the largest single

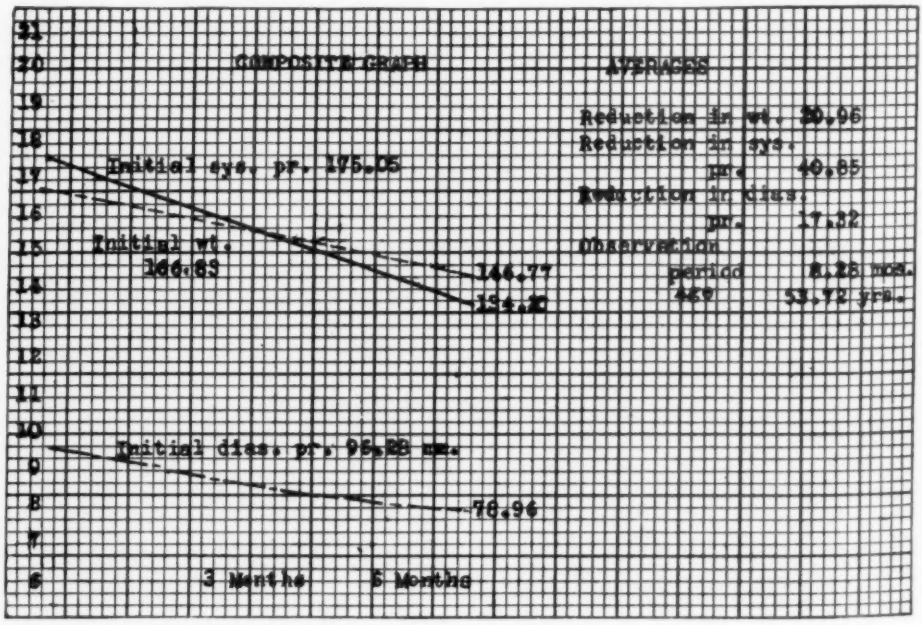

Chart 5. Graph of averages in 100 private patients personally observed at weekly to monthly intervals. Initial weight, 166.83 pounds. Reduced to 134.20 pounds over an average of 8.28 months. Pressure reduced from 175.05/96.28 to 134.20/78.96.

factor in the control of certain types of hypertension and the associated symptoms. It is more efficacious and more practicable than is generally appreciated.

5. One hundred private patients personally observed, many for long periods, showed the following average figures.

Initial weight	166.83 pounds
Initial systolic pressure	175.05 mm. Hg
Initial diastolic pressure	96.28 mm. Hg
Period of observation	8.28 months
Age	53.7 years
Reduction in weight	20.06 pounds
Reduction in systolic pressure	40.85 mm. Hg
Reduction in diastolic pressure	17.32 mm. Hg

6. In indigestion, gallbladder disease, and peptic ulcer, after middle life and especially with the tendency to hypertension, high caloric diets should be avoided.

7. Weight control after middle life presents one of the largest opportunities for personal prophylaxis and may be carried out in connection with the periodic health examination.

REFERENCES

Bauer, J.: Über Fettsucht, Wien. klin. Wchnschr., 1926, xxxix, 233-237.

Bauman, L.: Obesity: recent reports in the literature and results of treatment, Jr. Am. Med. Assoc., 1928, xc, 22-24.

Dublin, L. I.; Fisk, E. L., and Kopf, E. W.: Physical defects as revealed by periodic health examinations, Am. Jr. Med. Sci., 1925, clxx, 576-594.

Fisk, E. L.: The relationship of high blood pressure to other impairments, Am. Med., 1923, xxix, 446-454.

Foster, N. B.: The relations of hypertension to cardiorenal diseases, Am. Jr. Med. Sci., 1922, clxiv, 808-819.

Grafe, E.: Theorie und Praxis der Entfettungskuren, Med. Klin., 1929, xxv, 373-375.

Grafe, E., and Koch, R.: Über den Einfluss langdauernder, starker Übernährung auf die Intensität der Verbrennungen im menschlichen Organismus (Untersuchungen bei Mastkuren), Deutsches Arch. f. klin. Med., 1912, cvi, 564-591.

Kernohan, J. W.; Anderson, E. W., and Keith, N. M.: The arterioles in cases of hypertension, Arch. Int. Med., 1929, xliv, 395-423.

Kremer, D. N.: Obesity; observations on treatment by dietary measures, Ann. Int. Med., 1930-31, n. s. iv, 909-917.

Liebesny, P.: Die spezifisch-dynamische Eiweisswirkung, Biochem. Ztschr., 1924, cxliv, 308-350.

Master, A. M., and Oppenheimer, E. T.: A study of obesity: circulatory, roentgen-ray and electrocardiographic investigations, Jr. Am. Med. Assoc., 1929, xcii, 1652-1656.

Newburgh, L. H., and Johnston, M. W.: Endogenous obesity; misconception, Jr. Am. Dietet. Assoc., 1930, v, 275-285.

Preble, W. E.: Obesity: observations on one thousand cases, Boston Med. and Surg. Jr., 1923, clxxxviii, 617-621.

Rolly, F.: Zum Stoffwechsel bei der Fettsucht, Deutsche med. Wchnschr., 1921, xlvii, 887-889.

Rose, R. H.: Weight reduction—its indications in high blood pressure, Am. Med., 1923, xxix, 418-420.

Terry, A. H., Jr.: Obesity and hypertension. Jr. Am. Med. Assoc., 1923, lxxxi, 1283-1284.

Wang, C. C.; Strouse, S., and Saunders, A. D.: Studies on the metabolism of obesity. III. The specific dynamic action of food, Arch. Int. Med., 1924, xxxiv, 573-583.

An Evaluation of Stool Vaccines in Chronic Irritable Colon Therapy*†

By JOHN G. MATEER, M.D., F.A.C.P., and JAMES I. BALTZ, M.D.,
Detroit, Michigan

THERE are several conceptions regarding the etiology of the condition variously designated as chronic irritable colon, neurogenic mucous colitis, chronic non-ulcerative colitis, or mucous colopathy. Barker[1], Bockus[2], Jordan[3], Lichty[4], and the writer[5], have emphasized the importance of neurogenic factors in aggravating irritable colon symptoms, and the need for their control in any adequate program of therapy. Dorst and Morris[6] have recently emphasized the theory that chronic irritable colon is probably an allergic state, due to absorption of the foreign protein of certain stool bacteria. Others have contended that this condition is essentially a chronic infection, involving the wall of the colon, and that the term colitis can be properly applied, therefore, to this large group of cases.

Satisfactory therapeutic results can be obtained in a number of these cases from the employment of a comprehensive program, including the control of various neurogenic factors, bland diet, physiological regulation of the bowels, antispasmodics, and in some instances, changing the stool flora with lactodextrin and acidophilous milk. Certain obstinate cases, however, are resistant to the above types of treatment, and present an important therapeutic problem. Because of this fact, mainly, and also in view of the lack of agreement regarding the underlying etiology, it has seemed worthwhile to evaluate the practical effectiveness of vaccine therapy in these cases, and also to study the validity of the allergic theory. An abundance of material has been available, and it has therefore been possible to conduct our work upon a selected group of obstinate, irritable colon cases.

Only those autogenous stool vaccines were used, which produced definite, specific skin reactions with intradermal tests. Obviously this does not mean necessarily that the therapeutic results obtained were specific in character, viz., due to a direct effect of the vaccine upon the underlying etiological factor. *A priori*, the possibility of a non-specific mechanism, with a general, systemic effect, must be recognized.

METHOD AND ITS EVOLUTION

Warm stool specimens were cultured and vaccines prepared which con-

*Read before the American Gastro-Enterological Association, Atlantic City, May 5th, 1931.

†From the Gastro-Intestinal Division of Medical Department, Henry Ford Hospital.

tained originally one and one-half billion organisms per cubic centimeter. The vaccines were prepared according to the method of Hartman[7] and Jackson. In the more *recent* therapeutic experience with detoxified vaccines, detoxification has been carried out by subjecting viable, rather than heat killed, suspensions of the various strains to a concentration of one per cent purified and tested sodium ricinoleate for fifteen hours. The organisms were washed, then, several times with normal saline, to eliminate the sodium ricinoleate, and its irritating effect. Finally, trikresol (0.4 per cent) was added, as a preservative, and to provide sterilization.

Vaccine skin tests were carried out in *all* cases with *unaltered vaccine*, not subjected to sodium ricinoleate; and 1/20 c.c. of each vaccine was injected intradermally. These injections were made upon the flexor aspect of the forearm in all male patients, and upon the extensor surface of the thigh in female patients. The skin reactions were noted twenty-four hours later, as this seemed to be the optimum interval. The patients showing skin sensitivity were then started on vaccine injections of the one or more organisms to which they had reacted.

As a rule the first therapeutic dose of vaccine consisted of a total dose of one minim, containing the one or more organisms indicated, and injected subcutaneously. Subsequent injections were given at intervals of three to seven days, depending upon the degree of transient reaction of the patient to the last injection. In the earlier experience, the dose was increased about two minims each time. Since this increase, with a vaccine to which the patient was hypersensitive, led to a certain number of disagreeable reactions, it has been the custom for some months to increase the dosage not more than one minim per injection. Furthermore, if a patient had any appreciable reaction to this increase, the same dose was administered as was given at the previous injection, or a smaller dose. A course of treatments consisted of six to ten injections, with an average number of eight. The maximum dosage, therefore, consisted of eight minims. In some cases the dose could not be increased beyond two or three minims because of reactions. In fact, certain of the more sensitive patients were overwhelmed with the reaction following one minim of this concentrated vaccine.

More recently, therefore, more dilute vaccines have been prepared which contain from twenty-five to fifty million organisms per cubic centimeter. In some very sensitive individuals it has been necessary to provide further dilutions, even as low as ten thousand organisms per cubic centimeter, or less in occasional instances, before the vaccine was tolerated. In most cases, the symptomatic improvement is then definite. A longer course of vaccine injections, with very gradual increase of dosage, is given to these individuals. On the other hand, in those cases with greater tolerance for vaccine, better therapeutic results are often obtained when a somewhat more concentrated vaccine is used, provided the patient does not receive sufficient vaccine to produce a temporary reaction. The dosage should be adapted to the individual. If over-dosage is

avoided, a four-day interval between injections is very satisfactory. A shorter interval is tolerated by some patients. It is difficult to determine the most effective duration of the course of injections, and the desirability or practical necessity of attempting to obtain complete skin desensitization.

Until recently the unaltered vaccine has been used rather than vaccine detoxified with sodium ricinoleate, according to the recommendation of Dorst[6] and Larson[8]. The more recent experience with detoxified vaccine injected intramuscularly, is already sufficiently under way to indicate advantages. The resulting avoidance or diminution of temporary reactions seems to offer a distinct advantage, both from the standpoint of the patient's comfort, and from that of the therapeutic results obtained, this modification supplementing the effect of reducing the dosage. Certain patients have not tolerated the unaltered vaccine, but have been relieved greatly following treatment with the detoxified preparation.

Dorst's work was limited by giving vaccine therapy alone to a group of patients with chronic irritable colon, who had received no other type of treatment. It has not seemed justifiable in this work to withhold from these patients the older, comprehensive, therapeutic measures of recognized value, mentioned above. It is common knowledge that the majority of mild and moderate cases of irritable colon will become symptom-free with this program of therapy alone, provided sufficient care is taken in the elimination of the nervous factors.

In order to put vaccine therapy to a more severe test, and to eliminate favorable results not related to this type of treatment, we have utilized a carefully selected group of sixty-eight patients with chronic irritable colon, who had resisted the older therapeutic measures with definite persistence of symptoms. Vaccine therapy, of the type noted above, was introduced then as the only new therapeutic measure, other conditions remaining essentially constant. The practical results of such vaccine therapy upon colon distress, pylorospasm, and the associated symptoms are tabulated statistically in table I. The sixty-eight cases shown in this table have been treated with concentrated and unaltered vaccine. This

TABLE I
Practical Therapeutic Results of Autogenous Stool Vaccine Injections Upon Symptoms of 68 Cases*

	Colon pain	Pylorospasm syndrome	Constipation	Headache	Vertigo
Number of cases	57	20	34	23	11
Symptoms aggravated	2 (3%)	1 (3%)	0	2 (9%)	0
Symptoms unchanged	9 (16%)	8 (28%)	8 (24%)	8 (35%)	5 (46%)
Slight improvement	8 (14%)	3 (10%)	4 (12%)	1 (4%)	2 (18%)
Moderate improvement	11 (19%)	2 (7%)	4 (12%)	2 (9%)	0
Marked improvement	9 (16%)	2 (7%)	5 (14%)	4 (17%)	1 (9%)
Complete relief	18 (31%)	13 (45%)	13 (38%)	6 (26%)	3 (27%)
Total cases improved	46 (81%)	20 (69%)	26 (76%)	13 (56%)	6 (54%)

*Cases with persistence of symptoms after other therapy, showing results following introduction of vaccine therapy as the only new therapeutic measure. (Undiluted and unaltered vaccine was used in this group of cases.)

provides statistical data for comparison with the results obtained from diluted and detoxified preparations.

In the majority of obstinate cases thus treated, the pylorospasm and colon symptoms either improved or disappeared. Included in this group were a few unusually marked cases of pylorospasm, unassociated with colon distress, in which the symptoms had persisted in spite of comprehensive and intensive treatment. Yet these symptoms not infrequently disappeared in a striking manner with the introduction of vaccine therapy. Many of these cases noted marked improvement in their constipation, and were able to discontinue supplementary measures for the relief of this symptom, after vaccine treatment had been instituted. Certain patients, not listed in table I, but presenting a well established diarrhea as the main evidence of colon irritability, were also promptly relieved with vaccine. There were certain patients who had experienced for a long time very distressing headaches of the type often associated with constipation and gastrointestinal distress, not relieved by any of the previous therapeutic measures instituted, but which disappeared under vaccine therapy.

In this connection, it is admitted that in general the objective study of functional symptoms is difficult, and the danger of undervaluating psychological influence is recognized. However, this group of obstinate cases, whose symptoms had not responded satisfactorily to the previous therapeutic measures, did not represent an enthusiastic group, susceptible to therapeutic suggestion. Furthermore, symptoms as persistent as irritable colon or pylorospasm pain, or chronic headaches, are so stamped upon the individual as to be independent of any mental suggestion. A definite improvement or disappearance of such symptoms is therefore significant.

On the other hand, the symptoms referred to in table II are more purely subjective and less convincing as an index of the effectiveness of vaccine therapy. Even an objective change such as gain in weight is quite dependent upon improvement of appetite. Nevertheless, it was interesting to note that many of these patients, who had complained of chronic fatigability, were enthusiastic about their general improvement, and emphasized particularly the fact that they felt more energetic.

As to the average duration of symptomatic improvement, the above cases have been followed from one to twelve months. This period has been of sufficient duration to demonstrate the practical value of vaccine therapy.

Although vaccine therapy has proved very helpful in the majority of the more obstinate cases with irritable colon and related symptoms, there is evidence that it does not minimize the

TABLE II

Other Results of Vaccine Therapy in Group of 68 Cases

1. Appetite improved in 39 patients (57%).
2. Gain in weight in 11 patients (16%). (In these patients who gained weight, the average gain per patient was eleven pounds, and occurred with no change in dietary advice as previously outlined.)
3. Improvement in general feeling of well being occurred in 46 patients (68%). (Improvement very striking in some cases.)

therapeutic value or importance of the comprehensive therapeutic program, effective previously in the treatment of the majority of the mild and moderate cases. A major nervous problem which could not be controlled satisfactorily was an outstanding factor in some of the cases, whose gastrointestinal distress and pain were not relieved by vaccine therapy. In certain other cases, whose symptoms were relieved only partially by vaccine treatment, it was found that changing the stool flora was an extremely helpful supplementary measure. The importance of substituting a *diluted* vaccine, in those cases not relieved by a more concentrated preparation, has been emphasized.

Table III summarizes the frequency and degree of skin reaction occurring, with vaccines prepared from different stool organisms, in the total group of patients presenting chronic irritable colon and associated symptoms, upon whom skin tests were carried out. This group of 198 patients included the 68 treated with vaccine and referred to above, and 130 additional patients. The colon bacillus vaccine gave the most marked areas of skin reaction, although there was frequently marked skin sensitivity to the nonhemolytic streptococcus. Skin sensitiveness was less frequent, and usually less marked, when present, with the other organisms isolated. A number of patients were definitely skin sensitive to two or more organisms. The largest areas of skin reaction occurred with the *communior* strain of *B. coli*. In some cases, areas as large as nine or ten centimeters in diameter were seen, with associated injection of the lymphatic vessels leading to the axilla. In Table III the average intensity of the skin reaction, as evidenced by redness and swelling, has been measured on the scale of one to four plus for the various organisms.

In a sub-group of these cases, skin tests were done, both with these autogenous vaccines, and also with the corresponding strain of vaccines, originally prepared from stool cultures in other cases. It was found that the skin reactions in the majority of cases were similar with the autogenous and non-autogenous stool vaccines of the same organism. For example, with the two strains of colon bacillus, 98.5 per cent of the patients giving a positive skin test with the autogenous vaccine also gave a positive reaction with the

TABLE III

Frequency of Isolation from Stools, and Vaccine Skin Sensitiveness, of Common Stool Organisms (in 198 Patients Presenting Symptoms of Irritable Colon, Pylorospasm, or Both)

	Frequency of isolation from stool (cases)	Frequency of positive skin reaction when isolated (cases)	Average diameter and intensity of positive skin reactions
B. Coli (communis)	279 (91%)	170 (95%)	4.4 × 4.4 cm. + + +
B. Coli (communior)	77 (38%)	74 (96%)	4.5 × 4.5 cm. + + +
Streptococcus nonhemolytic	107 (54%)	67 (62%)	2.7 × 2.7 cm. + +
Staphylococcus aureus	27 (14%)	13 (49%)	2.4 × 2.4 cm. + +
Streptococcus viridans	11 (5%)	4 (36%)	2.8 × 2.8 cm. + +
Hemolytic streptococcus	2 (1%)	0	

corresponding nonautogenous preparation. In the case of the nonhemolytic streptococcus stool vaccines, the discrepancy between the skin reactions of autogenous and nonautogenous preparations was considerably greater than with the colon bacillus. This fact may be due to the great variety of strains of the streptococcus. There were *occasional* cases in which the nonautogenous vaccine gave a positive skin test, and the autogenous vaccine of similar strain produced a negative test.

A *control* group was carefully selected, which included twenty-one healthy individuals, with no present or past history of symptoms of irritable colon, pylorospasm, or any of the common allergic conditions. In comparing the findings in table IV with those of table III, it is noted that the frequency and degree of positive skin reactions are appreciably less in the control group than in the irritable colon cases. This is particularly true of the three common stool organisms, viz., the two strains of colon bacillus and the nonhemolytic streptococcus. The more infrequent stool organisms are probably chance findings. Their significance may lie in the fact that they come from the mouth, throat or gall tract. In this control group, positive skin tests occurred almost as frequently in the children as in the adults. The five children included were between five and fourteen years of age.

In table V the degree of skin reaction before, and two weeks after, completion of vaccine therapy was compared in a group of sixteen consecutive cases. It is noted that there was a definite tendency for the skin reaction to decrease in diameter and intensity. A decrease was noted in 75 per cent of the cases, including 12.5 per cent which exhibited a complete disappearance of the skin sensitivity, following vaccine treatment. As to the possibility of parallelism between symptomatic improvement and decrease of skin reaction following vaccine treatment, no final deductions can be drawn from this small group of sixteen cases. In the majority of cases there was some parallelism; but the significance of this finding was rendered questionable by the fact that, in one case, the skin reaction increased after vaccine, although there was marked symptomatic improvement; and, in a second case, there was a similar degree of symptomatic relief, with no change in the degree of skin

TABLE IV
CONTROL CASES
Skin Sensitivity of Twenty-One Normal Individuals, With no Present or Past Symptoms of Irritable Colon, or of any of the Common Allergic Conditions
(Sixteen Adults and Five Children)

Organisms	Frequency of isolation (cases)	Frequency of positive skin reaction when isolated (cases)	Average diameter and intensity of positive skin reactions
B. Coli (communis)	17 (81%)	11 (65%)	3 × 3 cm. + +
B. Coli (communior)	13 (62%)	9 (69%)	2½ × 3 cm. + +
Streptococcus (nonhemolytic)	14 (67%)	3 (21%)	1 × 1 cm. + +
Staphylococcus aureus	2(9½%)	1 (50%)	1 × 1 cm. +

Compare frequency and intensity of skin reaction to individual organisms in this control group with corresponding findings in irritable colon group (table III).

TABLE V
Comparison of Degree of Skin Reaction, Before and After Therapeutic Course of Eight Vaccine Injections (Unaltered Vaccine) (Sixteen Consecutive Cases)

Patients	Skin reaction *before treatment*	Skin reaction *after treatment*	Skin reaction *decreased**	Skin reaction *disappeared†*	Skin reaction *unchanged*	Skin reaction *increased*
1.	4 + (5 × 6 cm.)	2 + (3 × 4 cm.)	—			
2.	3 + (4 × 3 cm.)	2 + (3 × 3 cm.)	—			
3.	4 + (4 × 6 cm.)	3 + (3 × 4 cm.)	—			
4.	3 + (5 × 2 cm.)	3 + (5 × 3 cm.)			0	
5.	4 + (6 × 6 cm.)	2 + (3 × 3 cm.)	—			
6.	4 + (4 × 6 cm.)	1 +	—			
7.	3 +	No reaction		—		
8.	4 + (5 × 3 cm.)	1 + (2 × 1 cm.)	—			
9.	3 + (5 × 4 cm.)	2 + (3 × 2 cm.)	—			
10.	3 + (5 × 3 cm.)	2 + (2 × 2 cm.)	—			
11.	1 + (2 × 3 cm.)	3 +				+
12.	3 + (4 × 4 cm.)	3 + (4 × 4 cm.)			0	
13.	4 + (6 × 6 cm.)	4 + (6 × 7 cm.)			0	
14.	4 + (5 × 9 cm.)	3 + (4 × 4 cm.)	—			
15.	4 +	2 + (3 × 3 cm.)	—			
16.	3 + (3 × 4 cm.)	No reaction		—		
		TOTAL	10 cases or 62½%	2 cases or 12½%	3 cases or 19%	1 case or 6%

*Either decrease or disappearance of skin reaction in 75% of cases.
†Some skin sensitivity was still present after vaccine therapy in 87½% of cases.

sensitivity. This experience is not surprising, however, in view of the existing knowledge regarding marked changes in degree of skin sensitivity, which occur occasionally in various types of allergy, without any treatment having been administered.

In almost every instance where vaccine therapy is used with good effect, the question is raised as to whether one is dealing with a specific effect, or with a nonspecific protein reaction. This attitude is due mainly to the excellent work of Peterson[9], Joseph L. Miller[10], and others, who have demonstrated the occurrence of nonspecific protein therapy, its value in various conditions, and the importance of controlling any work that appears to show specific vaccine effects.

As regards the nature of the therapeutic action of autogenous stool vaccine treatment in irritable colon and pylorospasm patients, the evidence favoring a specific effect is:

1. That there is a marked skin reaction to certain of the isolated stool bacteria in a very high percentage of this irritable colon group of patients;

2. That the best therapeutic results in relieving irritable colon pain seem to occur when systemic reactions are avoided, by using small, rather than large, doses, and by giving the detoxified vaccine; whereas Miller[10] and others have emphasized the im-

portance of obtaining a systemic reaction, preferably with some fever, if satisfactory results are to be obtained from a nonspecific type of therapy.

On the other hand, the main point of evidence suggesting a nonspecific protein mechanism is that an appreciable percentage of the *control* group shows skin sensitivity to the common stool organisms. For example, 69 per cent of this group were skin sensitive to the *B. coli communior* vaccine, when this organism was isolated.

When one considers the fact that the human race is exposed universally and throughout life to most intimate contact with, and absorption of, disintegrating stool organisms, it is not surprising that an appreciable percentage of the control group was skin sensitive to stool vaccines. It is also a well recognized fact that there are individuals who have never had hay fever symptoms, who, nevertheless, show skin sensitivity to ragweed. This fact, in itself, is not supportive of either specificity or nonspecificity. When the real mechanism of allergy and immunity is discovered, questions of this sort will be answered more definitely.

Recently we have started to treat a control group of irritable colon patients with nonspecific, milk protein injections, as a further check upon the exact mechanism underlying the above therapeutic results in irritable colon cases; but this work is too recent to discuss results at the present time. We are also studying a group of irritable colon patients from the standpoint of their skin sensitivity to various other substances, in an effort to further elucidate the problem.

SUMMARY

1. Autogenous, stool vaccine therapy, utilizing organisms to which the patient is skin sensitive, has been employed in the treatment of a group of patients with obstinate irritable colon and associated symptoms. In order to put vaccine therapy to a more severe test, and to eliminate favorable results not related to vaccine treatment, a carefully selected group of sixty-eight patients has been utilized, who had resisted previously a comprehensive program of the older therapeutic measures with definite persistence of symptoms. Vaccine therapy was then introduced as the only new therapeutic measure, other conditions remaining essentially constant.

2. The most important fact revealed in the above observations has been the demonstration of the definite therapeutic value of properly selected stool vaccines, in partially or completely relieving the remaining symptoms of the majority of these more obstinate irritable colon patients. The above experience has demonstrated that vaccine therapy not only tends to relieve the colon distress and associated symptoms, but also makes it possible for a majority of these patients to tolerate a greater variety of diet and a greater degree of nervous activity, and furthermore, to place less emphasis upon the supplementary measures for the control of constipation.

3. The therapeutic results tabulated statistically in table 1 were obtained with concentrated and unaltered vaccine, giving an average course of eight injections. The results obtained more recently, with a longer course of more dilute vaccines detoxified with sodium

ricinoleate, and with greater emphasis upon determining the optimum dosage for each individual, have been even more satisfactory than those outlined in table I, and obtained with the original method. The exact statistics with the newer method have not yet been compiled; but certain patients who failed to tolerate the concentrated, unaltered vaccine have obtained symptomatic relief from the more recent method.

4. Vaccine therapy *should not displace* the older therapeutic measures of recognized value in irritable colon cases, such as control of various neurogenic factors, proper diet and regulation of bowels, the discriminate use of antispasmodics, and, in some cases, the changing of stool flora. This comprehensive program, with special attention to correction of all faulty habits, should precede vaccine therapy for the following reasons:

(A) The symptoms of the majority of the mild and moderate cases will disappear with this comprehensive therapy. Vaccine therapy is not necessary, therefore, in this group.

(B) In the obstinate cases, whose symptoms do *not disappear* with the comprehensive program, the colon irritability and spasm, nevertheless, tend to be *partially* reduced by the elimination of aggravating neurogenic factors and other faulty habits. If vaccine therapy is introduced subsequently, as a supplementary measure, the end results of colon therapy, as well as the general condition of this group of patients, will be more satisfactory than if the comprehensive therapy were to be omitted, and vaccine therapy alone utilized. As the effects of vaccine therapy become apparent, those measures in the comprehensive program which are rendered unnecessary should be discontinued.

5. The colon bacillus was the most frequently isolated organism, and, as a vaccine, gave the most frequently positive and most marked skin reactions. (See table III.)

6. Skin sensitivity in a *control* group of healthy, asymptomatic individuals, with no history of any irritable colon or allergic symptoms, was tested. (See table IV.) Sixty-five per cent of the control group showed skin sensitivity to one strain of the colon bacillus, and sixty-nine per cent to another strain.

7. Skin sensitivity to stool vaccines was shown to decrease and even to disappear, in some irritable colon cases, under vaccine therapy. (See table V.)

8. As to the exact mechanism involved in the production of the above therapeutic results of vaccine therapy, evidence is presented for and against a specific vaccine effect. The available evidence does not justify a definite decision upon this question. A control group of patients is being treated with nonspecific milk-protein injections.

REFERENCES

[1]Barker, L. F.: On the management of the spastic colon and mucous colopathy, especially in hypervagotonic persons, Am. Jr. Med. Sci., 1929, clxxviii, 606-615.

[2]Bockus, H. L.; Bank, Jos., and Wilkinson, S. A.: Neurogenic mucous colitis, Am. Jr. Med. Sci., 1928, clxxvi, 813-829.

[3]Jordan, S. M.: The irritable colon, Jr.

Am. Med. Assoc., 1929, xciii, 592-595.

[4] LICHTY, J. A.: The present consideration and care of the colon, Jr. Am. Med. Assoc., 1931, xcvi, 649-653.

[5] See discussion of Dr. Lichty's paper.

[6] DORST, S. E., and MORRIS, R. S.: Bacterial hypersensitivity of the intestinal tract, Am. Jr. Med. Sci., 1930, clxxx, 650-656.

[7] HARTMAN, F. W., and JACKSON, E.: Isolation, selection and preparation of bacteria for vaccines, to be published in Am. Jr. Clin. Path. In press.

[8] LARSON, W. P., and EDER, H.: Immunization against diphtheria with toxin detoxified with sodium ricinoleate, Jr. Am. Med. Assoc., 1926, lxxxvi, 998-1000.

[9] PETERSON, W. F.: Protein therapy and non-specific resistance, MacMillan Co., New York, 1922.

[10] (a) MILLER, J. L.: Non-specific therapy, Medicine, 1927, vi, 513-549. (b) MILLER, J. L.: The present status of non-specific therapy, Trans. Med. Sec., Jr. Am. Med. Assoc., 1930, vol. 95, 464-468.

On the Advantages of Being Ill

"TO BE ill, or to undergo an operation, is to be initiated into the mystery of nursing, and to learn the comforts and discomforts of an invalid's life; the unearthly fragrance of tea at daybreak, the disappointment of rice pudding when you thought it was going to be orange-jelly, and the behavior of each constituent part of the bedclothes. You know, henceforth, how many hours are in a sleepless night; and what unclean fancies will not let us alone when we are ill; and how illness may blunt anxiety and fear, so that the patient is dull, but not unhappy or worried; and how we cling to life, not from terror of death, nor with any clear desire for the remainder of life, but by nature, not by logic. In brief, you learn from your own case many facts which are not in text-books and lectures; and your patients, in the years to come, will say that they prefer you to the other doctor, because you seem to understand exactly how they feel. I wish you therefore, young man, early in your career, a serious illness, or an operation, or both. For thus, and thus alone, may you complete your medical education, and crown your learning with the pure gold of experience. The crown of experience is like the crown of Lombardy, a band of iron set in a band of gold: and it is believed, even now, by some people, that the iron of that crown is mors valuable than the gold."—From *Confessio Medici*, by STEPHEN PAGET, F.R.C.S. (The Macmillan Company, New York City, 1931, Reissue.)

Diagnostic Value of Secretory Function in Gastric Disease: Various Methods Studied and Compared*†

By Ernest H. Gaither, M.D., F.A.C.P., *Baltimore, Md.*

During the past few years, renewed and continued interest has been manifested by numerous investigators in the domain of gastric secretion, and the test which has apparently met with almost universal favor has been that in which histamine, subcutaneously administered, has been used as the stimulator of gastric gland activity.

A careful survey of the current literature on this subject reveals the interesting fact that practically all observers agree as to the direct action of this drug upon the gastric glands, or upon a mechanism which has intimately to do with their secretion; innumerable tests of acidity have satisfied many workers in this field that a study of the acid values by means of the histamine test, will enable one to form an excellent and reliable idea as to the ability of the glands to secrete acid, and will furnish information of true diagnostic worth.

Immediately after the successful application of the histamine test for acid determination, certain investigators entered upon a searching inquiry into the matter of enzymes, chlorides, and other constituents; and, as was to be expected, conflicting claims, either hostile or extravagantly favorable in nature, were made as to the dependability and diagnostic value of the test in the presence of disease under varying conditions. Because of these conflicting and unsatisfactory views, it was decided to confine the present investigations to a consideration of acid values only.

The proponents of the histamine test state that its advantages are not only the subcutaneous application, which eliminates the disturbing psychic factor induced by the mastication of solids and the swallowing of liquids, as in other test meals; but also the fact that the dosage may be standardized and pure juice obtained. Then, too, because of the direct action of this substance on the gastric glands, many cases which show achylia by other test meals will give an acid response after the injection of histamine.

Objections to the Ewald, alcohol, and various other test meals, have been presented by some observers, who claim that contamination and dilution

*Read before the American Gastro-Enterological Association, Atlantic City, May 5, 1931.

†From the Gastro-Intestinal Clinic of Johns Hopkins Hospital, Baltimore.

of the gastric content are so engendered by the introduction of these substances, as to mask the true secretory power of the gastric glands; they aver, also, that the neutralization of the hydrochloric acid by saliva, food, and regurgitation of duodenal contents further complicates an already deceptive result. The contention is advanced that tests other than the histamine injection do not provide sufficient stimulation to obtain an adequate response from the gastric glands.

I am unable to accept these objections as valid; some of them would appear to be mere sophistry. For instance, it can certainly be proven that in the majority of cases, bread and water supply an adequate stimulation to gastric gland secretion; and as for the argument regarding neutralization by swallowed saliva and regurgitation of duodenal contents, as well as that concerning contamination brought about by solids and liquids, does not such a process more nearly simulate the usual physiologic routine of digestion? And after all, are we not endeavoring to ascertain the functional capacity of the gastric glands under conditions as nearly physiologic as possible? No one can gainsay the fact that Nature never depends upon histamine injections to bring about a normal response of the gastric secretory glands.

This study represents an investigation by three methods, using the fractional extraction.

I. The Alcohol Test Meal

Technique. Patient given fifty c.c. of an 18 per cent alcohol solution containing 0.005 gm. phenolphthalein.

II. Ewald Meal

Technique. One slice of white bread without butter, and one glassful of cool water, ingested forty-five minutes before extraction.

III. Histamine Test

Technique. Dissolve one histamine tablet of 0.005 gm. in three c.c. distilled water; multiply the weight of the patient by 0.005; this gives the number of c.c. of histamine solution to be administered subcutaneously.

In estimating acidities we made use of the accepted figures of twenty to forty degrees for free hydrochloric acid, and forty to sixty degrees for total acidity, as indicating normal limits, realizing that the acidities following histamine stimulation are considerably higher.

This would seem the opportune moment for stressing two important points; first, in selecting this series of cases for research, every available method of investigation was brought into use in order to insure the absolute correctness of each diagnosis; second, we were not prejudiced for or against any of the methods, and were eager to consider all evidence and all established facts judicially and in order, so as to reach if possible, a correct and unbiassed conclusion as to the diagnostic value possessed by any of the methods.

The cases were grouped under the following headings:

1. Achylia gastrica
2. Adhesions (postoperative; plastic gastric operations)
3. Colitis
4. Gastric neurosis

5. Hepato-biliary pathology (cholecystitis; cholelithiasis)
6. Ptosis and atony
7. Peptic ulcer

Achylia Gastrica

We charted in this group all those cases showing in any of the tests a lack of free hydrochloric acid, and have included all such cases from the various groups. There were nineteen in this group.

8 Achylia by all three tests.
6 Achylia by alcohol; normal or hypochlorhydria by histamine and Ewald.
3 Achylia by alcohol and Ewald; normal or hypochlorhydria by histamine.
1 Achylia by histamine and alcohol; hypochlorhydria by Ewald.
1 Achylia by histamine and Ewald; hypochlorhydria by alcohol.

19 Total.

These results indicate that a small majority of cases showing achylia by the Ewald and alcohol tests will present free hydrochloric acid after histamine stimulation.

Adhesions (Post Operative)

There were five in this group.

4 Hyperchlorhydria by all three tests.
1 Achylia by alcohol; normal by Ewald and histamine.

5 Total.

In this group the histamine shows no diagnostic advantage over the Ewald and alcohol tests.

Colitis

Six were included in this group.

2 Hyperchlorhydria by all three tests.
1 Normal acidity by all three tests.
1 Moderate hyperacidity by histamine; normal by Ewald and alcohol.
1 Hyperacidity by histamine; achylia by Ewald and alcohol.
1 Hypochlorhydria by histamine and Ewald; achylia by alcohol.

6 Total

In this group there is certainly no outstanding advantage of the histamine test.

Gastric Neurosis

There were twenty-five in this group.

10 Hyperacidity by all three tests.
10 Hypochlorhydria and normal by all three tests.
2 Hyperacidity by histamine; normal or moderate hypochlorhydria by alcohol and Ewald.
2 Achylia by histamine, normal by alcohol and Ewald.
1 Achylia by Ewald and alcohol; hypochlorhydria by histamine.

25 Total.

This survey shows that in the great majority of cases all three fractional studies coincide. The labile character of gastric secretion is emphasized.

Hepato-Biliary Pathology

Twelve in this group.

5 Hyperacidity by all three tests.
2 Achylia by all three tests.
1 Hypochlorhydria by all three tests.

1 Achylia by alcohol; hypochlorhydria by histamine and Ewald.
1 Normal by alcohol; hyperchlorhydria by histamine and Ewald.
1 Normal by Ewald; hypochlorhydria by alcohol; moderate hypochlorhydria by histamine.
1 Normal by histamine and Ewald; achylia by alcohol.

12 Total.

The histamine presents no diagnostic advantage in this group.

Ptosis and Atony

Twelve in this group.

1 Achylia by all three tests.
6 Hyperchlorhydria by all three tests.
2 Hyperchlorhydria by histamine; normal by Ewald and alcohol.
1 Normal by histamine; achylia by alcohol; hypochlorhydria by Ewald.
1 Normal by alcohol; achylia by Ewald; hyperchlorhydria by histamine.
1 Achylia by alcohol; hypochlorhydria by histamine and Ewald.

12 Total.

Histamine here gives no advantage from a diagnostic standpoint.

Peptic Ulcer

Thirty in this group.

20 Hyperchlorhydria by all three tests.
1 Normal by all three tests.
1 Hyperchlorhydria by histamine and Ewald; hypochlorhydria by alcohol.
1 Achylia by histamine and alcohol; hypochlorhydria by Ewald.
1 Achylia by Ewald; normal by alcohol; hyperchlorhydria by histamine.
2 Normal by alcohol and Ewald; hyperchlorhydria by histamine.
1 Normal by Ewald and histamine; hypochlorhydria by alcohol.
1 Normal by alcohol and histamine; hypochlorhydria by Ewald.
1 Normal by alcohol; hyperchlorhydria by histamine and Ewald.
1 Normal by Ewald; hypochlorhydria by alcohol; hyperchlorhydria by histamine.

30 Total.

The superiority of histamine is certainly not proven in this group of cases.

Summary

This investigation was instituted for the purpose of determining whether the histamine test is possessed of outstanding superiority over other methods of estimating the acid values in gastric secretion.

One hundred cases were selected—a cross-section of dispensary patients—and in these cases every available method was used in order to insure proper diagnosis. In each case three tests—histamine, Ewald, alcohol, using the fractional method—were applied.

The results so obtained are tabulated and reviewed, with the following conclusions drawn:

1. The Ewald and alcohol meals are effective stimulators of gastric secretion.

2. The objection that the swallowing of saliva, the contamination and neutralization of gastric juice by the meal itself, and also the regurgitation of duodenal contents, markedly or ef-

fectually mask the results of these meals *is not sustained*.

3. The labile character of gastric secretion is proved.

4. Histamine does in a number of cases offer an advantage in establishing the fact that the glands do possess the power of acid secretion when after the Ewald or alcohol test an achylia would seem to be present.

5. The alcohol meal is not nearly so potent a stimulant of gastric gland secretion as the Ewald meal.

6. There is ample justification for the continuance of the Ewald meal as a dependable test for gastric gland secretion.

7. The claim that histamine is vastly superior to the Ewald meal as a stimulant of acid secretion of the gastric glands has been disproved.

8. It would really appear that the application of bread and water as a test of gastric gland secretion is more physiologic than the subcutaneous application of the foreign body, histamine, and that it presents a result more in keeping with normal bodily economy.

I wish to add a note of appreciative thanks for very valuable assistance so ably rendered by Miss Stearns, Miss Lesser, and Miss Kravetz, of the Gastro-Intestinal Clinic of the Johns Hopkins Hospital in making the tests used in this investigation.

The Clinical Significance of Choroidal Tubercles*

By R. T. Paton, M.D., *Baltimore, Md.*

BEGINNING about four years after the discovery of the ophthalmoscope we find descriptions of choroidal tubercles in the medical literature. Among the investigators who are associated with these early observations are Jaeger, Von Graefe, Leber, Manz, Bouchert, Gerlach, Fraenkel, and Horner. These descriptions included the two types commonly known as (1) the granuloma or solitary tubercle, (2) choroidal miliary tubercles. The former is usually associated with chronic tuberculosis, and the latter with acute miliary tuberculosis. These observations have been substantiated by later investigators.

Studies of pathological material have shown that the usual number of tubercles in one eye is three or four, but in some cases there may be a much larger number. Morton reported as many as 70; Parsons, 60-70; Cohnheim, 52, and Boch, 49. The size varies from 0.4 mm. to 5 mm. Groenouw stated that tubercles smaller than 0.6 mm. could not be seen with the opthalmoscope.

Some observers claim that miliary tubercles are frequent in general miliary tuberculosis. Cohnheim found them in every instance in the 18 patients he examined, Boch in over 80 per cent, and Carpenter and Stephenson in 50 per cent; while Marple on repeated and careful examination found the appearance of tubercles to be the rule and not the exception. Marple's earlier investigations gave a very small percentage, but this error he discovered was due to failure to make frequent examinations, especially in the late stages of the disease.

Groenouw in a series of cases, including 378 cases of miliary tuberculosis and tuberculous meningitis, found tubercles of the choroid present in 35 per cent of the cases examined either during life or at autopsy. In 222 cases of the series tuberculous meningitis was the clinical and pathological diagnosis; 44 per cent showed tubercles of the choroid. In contrast with these statistics are the figures given by Bredech. Bredech, 1916, after examining the evidence presented by reports, in which careful examination of the fundus had been made, collected a total of 226 cases of tubercular meningitis, in which 14.6 per cent showed tubercles of the choroid. No doubt these percentages would have been somewhat higher had pathological studies been made in every case. He thought that the number would have reached 20 per cent had there also been more frequent examination of the eyes.

*From The Fifteenth Annual Clinical Session at The Wilmer Ophthalmological Institute, of the Johns Hopkins University and Hospital, Baltimore, Md., March 26, 1931.

The diagnosis of choroidal miliary tubercle is dependent to a large extent upon the rapid and unexpected appearance of the tubercle. This fact has been stressed by Fraenkel, Groenouw, Lotin, Weis, and many others. Marple reported a case in which an ophthalmoscopic examination, made at 6 P.M., did not reveal the presence of a tubercle, but four hours later a tubercle appeared. Moore reported one developing in five hours, while Strider and Weis saw tubercles develop in 12 hours. The discrepancy among various authors as to the existence of tubercles, especially in meningitis, is probably due to two factors: (1) Difficulty in examination—tubercles in these cases are usually found only in the terminal stages. (2) Failure of repeated and careful examination. Tooke reported choroidal miliary tubercles in one case as early as 33 days, but in the majority of cases they did not appear until about three days before death. The clinical examinations in all these cases were verified by post mortem examinations.

Both eyes should be examined. Cohnheim found tubercles in both eyes in 15 out of 33 cases examined. These observations were verified by autopsy and microscopic study.

The presence of miliary tubercles of the choroid is often a valuable aid before the laboratory tests have been completed, in making a diagnosis in cases suspected of being typhoid fever, meningitis or miliary tuberculosis. Series of cases, in which the value of the ophthalmoscopic examinations is proved, have been published by Bollach, Hillernan and Laporte. Van der Hoeve reported a case of mastoiditis with cerebral symptoms, in which the existence of military tuberculosis had not been suspected until the ophthalmoscopic examination revealed the presence of miliary tubercles. The diagnosis was confirmed at autopsy.

The prognosis in miliary tubercles of the choroid is nearly always bad. Usually there is a meningitis. Jessup, in a series of 15 cases, in which there were tubercles of the choroid present, found that 14 had tuberculous meningitis. Only one case was free from meningeal complications. The clinical diagnoses were confirmed by post mortem examinations.

Solitary or conglomerate tubercle of the choroid is a rare and destructive disease, and is probably always secondary to tuberculous disease in other parts of the body. The affection is usually unilateral, though bilateral incidence has been reported and verified by post mortem studies. The condition is usually seen in the early years of life, but its occurrence has been reported as late as 62 years (Nedden).

Differential diagnosis is often difficult, for we have to distinguish the tubercles from the various malignant growths, such as glioma, retinoblastoma, detachments with massive exudates, etc. In some cases the diagnosis can be made only after the eye has been removed. If the diagnosis is made, enucleation is not indicated unless all therapeutic measures, especially tuberculin, which in recent years has appeared to act favorably in many cases, have failed.

The results of the study of a case of miliary tubercles, which was recently observed, may be of interest.

The patient, a 10 year old boy, was ad-

mitted to the Harriet Lane Home 12 days after the onset of the illness. There was no history of tuberculous contact. On admission the patient was acutely ill. The chief symptoms were headache, nausea, vomiting, drowsiness and mild diarrhea. The boy was temporarily isolated and treated as a case of typhoid. The eye examination made on admission gave the following results.

There was slight suffusion of the conjunctiva, dilated pupils, and slight photophobia. The patient's general condition became progressively worse. Laboratory tests at this stage gave practically negative results. These tests were repeated two days later. Lumbar puncture showed 290 cells per c.c.; fluid was under pressure, and a pellicle was formed on standing. The ophthalmoscopic examination revealed slight bilateral optic neuritis with subretinal exudate forming adjacent to the papillae. The veins were engorged and there was definite retinal edema extending out toward the periphery. The diagnosis of tuberculous meningitis was established on the following day, 16 days after the onset of the illness, when tubercles were visible in both eyes. Ocular examinations had been made three or four times a day, so that it is not likely that tubercles had formed earlier in the course of the disease. The patient died two days later. The pathological diagnosis finally established was miliary tuberculosis with meningitis. Tubercles were formed in practically all organs of the body.

BIBLIOGRAPHY

JAEGER, E.: Über Choroidealtuberkel, Oesterr. Ztschr. f. Prakt. Heilk., 1855, Jahrgang, i, 9-10.

VON GRAEFE, A., und LEBER, T.: Über Aderhauttuberkeln, Arch. f. Ophth., 1868, xiv, 183-206.

MANZ, W.: Tuberculose der Choroidea, Arch. f. Ophth., 1858, iv, Part ii, 120-126.

BOUCHUT, M.: Des tubercles de la choroide et de la retine pouvant servir au diagnostic de la tuberculose cérébrale, Gaz. des Hôp., 1869, ii.

FRÄNKEL, B.: Weitere Beobachtungen von Aderhauttuberkeln, Berl. klin. Wchnschr., 1869, vi, 37-39.

HAAB, O.: Die Choroiditis tuberculosa chronica, Arch. f. Ophth., 1879, xxv, 221-236.

PARSONS, J. H.: Diseases of the eye, 1930, Macmillan Company, New York, 530.

COHNHEIM, J.: Über Tuberculose der Choroidea, Virchows Arch. f. path. Anat., 1867, xxxix, 49-69.

GROENOUW, A.: Beziehungen der Allgemeinenleiden und Organerkrankungen zu Veränderungen und Krankheiten des Sehorganes, Handbuch der Augenheilkunde, 1920, Dritte Auflage, 1074-1087.

CARPENTER, G., and STEPHENSON, S.: A case of tubercle of the choroid presenting certain clinical and pathological peculiarities, Ophthalmoscope, 1905, iii, 374-380.

MARPLE, W. B.: Tubercle of the choroid in tuberculous meningitis, Ophthalmoscope, 1912, x, 559-563.

BREDECK, J. K.: The choroidal tubercle in tuberculous meningitis, Am. Jr. Ophth., 1915, xxxiii, 1-8.

LOTIN, A.: Über die tuberkulose disseminierte Choroiditis und über die Miliartuberkeln der Aderhaut, Klin. Monatsbl. f. Augenh. 1925, lxxiv, 262.

WEIS, L.: Über die Tuberkulose des Auges, Arch. f. Ophth., 1877, xxiii, 57-158.

TOOKE, F.: Tuberculous meningitis with special reference to the tubercle of the choroid and its pathological manifestations, Tr. Am. Ophth. Soc., 1915-16, xiv, 218-232.

BOLLACK, J.; HILLEMAND, P., and LAPORTE, A.: Tuberculose miliaire de la choroide, Annales d'Oculistique, 1927, clxiv, 660.

VAN DER HOEVE, J.: Examen du fond de l'oeil dans la tuberculose miliaire, Bull. Soc. de pédiat. de Paris, 1925, xxii, 511-514.

JESSOP, W. H.: A discussion of intraocular tuberculosis, Brit. Med. Jr., Sect. Ophth., 1925, ii, 430-433.

RANDOLPH, R. T., and SCHMEISSER, H. C.: A clinical and pathological study of two cases of miliary tubercles of the choroid, Tr Am. Ophth., 1915-16, xiv, 585-592.

Laboratory Methods in Clinical Medicine*

Report of Three Cases

(1) Diaphragmatic Eventration
(2) Chronic Nephritis without Hypertension, Cardiac Hypertrophy or Retinal Changes
(3) Unusual Case of Cholelithiasis

By SAMUEL LEVINE, M.D., *Brooklyn, N. Y.*

FROM time to time the statement is made at medical meetings and in the medical press that the introduction of laboratory methods in the practice of medicine has impaired the physician's powers of observation and his clinical sense; and that, as a result of this, his diagnostic abilities have deteriorated and both patient and physician are thereby the losers.

It must be admitted that there is some truth in this statement. The keen sense perceptions of the animal, so important in its struggle for existence, are very much diminished in civilized man (Darwin[1]). In his struggles he has developed tools of far greater effectiveness than instincts and sense organs. As a result, these are used much less and have therefore lost some of their power. The fact, however, is that man with his duller perceptions has conquered the animal kingdom; and that portion of mankind that wields the technical power of Western civilization dominates all those races that still retain the primal keenness of their senses.

Formerly, the physician, in the practice of his art, was forced to rely upon his senses only, which were consequently developed to a fine degree. But this was due to necessity, not to choice. At present, more accurate methods have replaced them to a certain extent. The thermometer registers changes in temperature much better than the thermesthetic sense; the hemoglobinometer and hemacytometer are much more reliable in the study of blood diseases than the naked eye; and general clinical impressions based upon empirically acquired knowledge have given way to tests based upon scientifically controlled facts. It is doubtful whether these have already resulted in dulling physicians' senses. That the progress of medicine along present lines may have such an effect is very likely. The senses of the physician are no exception to the biologic laws of use and disuse (Darwin[2]). Nor is this fact to be deplored any more than the general lessening of sense acuity in the entire human race, particularly in its civilized portion.

The following cases illustrate the

*Received for publication, June 5, 1931.

value of laboratory methods in clinical medicine.

Diaphragmatic Eventration

History. Mr. N. S., aged 31, automobile mechanic, came to my office Nov. 15, 1927. His wife and three children were alive and well. He did not use alcohol and smoked twelve to fifteen cigarettes a day. He had had the usual diseases of childhood. At the age of ten, he was very ill and was confined to bed for a year. The late Dr. Robert Schultz, who treated him during that illness, informed me that, "When ten years of age, Mr. S. was ill with abdominal cramps, diarrhea and vomiting. He lost a lot of weight. Diagnosis was tuberculous peritonitis. He was confined to bed for one year and made a good recovery."

Present Illness. In the preceding July, during lunch hour, he was standing in front of the garage where he was employed. The car on which he had been working that morning was raised on a jack in the street near the sidewalk. He saw a truck come along and strike the car, throwing it off the jack. No one was injured. "I said nothing, but in the evening, I felt faint and something was pumping in the left side of my chest." Here he pointed to precordium and left axilla. "I had difficulty in breathing and developed a crampy feeling in my left armpit." Then the following symptoms developed: heartburn, relieved by bicarbonate of soda, not relieved by food; sour eructations, and abdominal cramps after meals. There was numbness in left upper and lower extremities, and in front and behind left ear. He had nightmares and dreamt he was falling from some great height. Patient had to give up his position and went to the country for a rest, without any improvement in his condition.

Examination. The patient was a medium-sized, hairy male. He had two accessory rudimentary mammae on each side, below and to the inner side of the normally placed mammae, undeveloped and adherent lobules of both ears, and a remarkably large penis. The pulse rate was 72. The blood pressure was 126 systolic and 60 diastolic in both arms. His weight was 125 pounds (56.8 Kg.). Dull tympany was present below left scapula with diminished vocal fremitus and absent breath sounds. After radiographic examination and when *purposely looked* for, the following were noticed: When stomach was empty, there was dull tympany at the base of the left lung and the heart was only slightly displaced to the right. When the stomach was full, the heart was much more displaced to the right (Assman[3]), and the base of the left lung was flat. Splenic dullness could not be elicited because of marked tympany in that area. The left lower chest was flattened, more so over precordium. While the chest expanded with inspiration in a normal manner, the intercostal spaces retracted, particularly over precordium.

Radiographic examination showed that the left dome of the diaphragm was high and moved slightly downward with inspiration. The heart was displaced to the right (figure 1). Barium gruel was then administered. Fluoroscopy of stomach revealed that the greater part of it was situated under the lower left ribs. The air bubble was large. The fundus rested against the diaphragm. Body and pylorus were situated in the left side of abdomen. The incisura angularis was absent and the pyloric part ran straight downward. The duodenum was pulled over to the left. The stomach emptied rapidly. (Figure 2).

A barium enema was given and it was noticed that the hepatic flexure of the colon formed an obtuse angle and the transverse colon ran upward and to the left (figure 3).

Cholecystograms taken after the administration of tetra-iodophenolphthalein by mouth, revealed the gallbladder faintly outlined and with poor concentration. This was probably due to the abnormal position of the duodenum which interfered with the normal function of the papilla duodeni.

Proctoscopic and sigmoidoscopic examinations were negative. Urinalysis was negative. Two out of three specimens of feces gave a faintly positive reaction to the benzidine test. Blood examination was negative except for an increase in the icteric index which was 10.7, probably caused by the displacement of the duodenum which interfered

with the patency of the common bile duct. The blood Wassermann reaction was negative and the blood count was normal.

Comment

This man was seen by several good clinicians, who, in view of the history and symptoms made a diagnosis of neurosis. Some of them also thought that he had a lesion in the left lung.

These patients are usually diagnosed as neurotics and psychopaths. Friedrich Schneider in 1900, the first man with diaphragmatic eventration to be studied with the roentgen rays told Dr. Hirsch[4]: "Many doctors did not believe that I suffered". Leichtenstern's[5] patient with diaphragmatic hernia was considered by him as a "simulant", a neurotic, and he really acted as such. This has also been the experience of Carman and Fineman[6], Soresi[7], Funk[8], and Funk and Manges[9]. Walton[10] writes, "During the past ten years, there have been more authentic cases of eventration reported than in the previous 139 years, when Petit first reported his case." "Clinically", says Hitzenberger[11], "this diagnosis can only be surmised; without the aid of the roentgen rays, it can hardly ever be made." During eight years in a very active radiographic

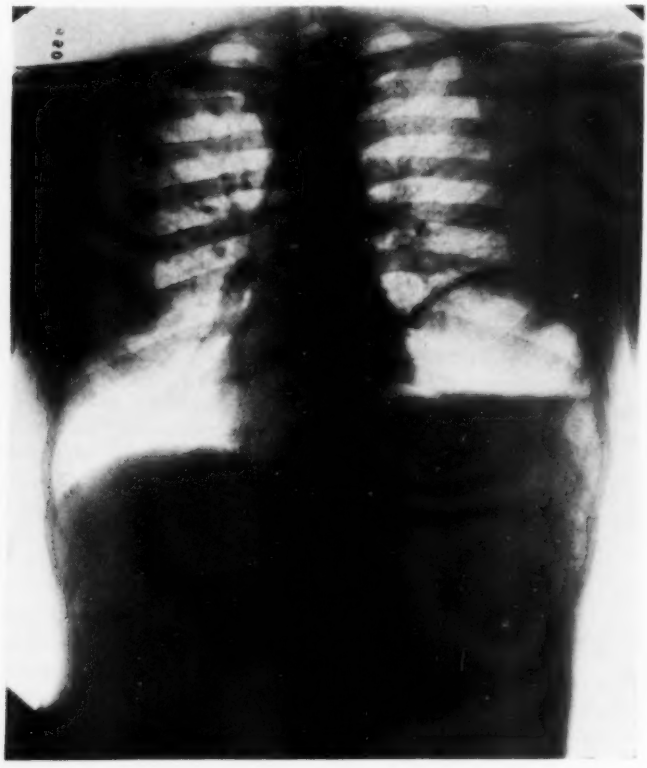

Fig. 1. Case 1. Left dome of diaphragm high. Heart displaced to the right. This film was made one hour after dinner.

service he saw twenty cases of eventration. So many cases have been reported in recent years that Hitzenberger thinks it is fortunate that not all observed cases are published.

It is of real clinical importance to recognize this condition because a mistaken diagnosis may have serious consequences. Since the physical signs in the lung strongly suggest the presence of fluid, a needle may be introduced which would enter the stomach with, perhaps, fatal results (Pepper[12], Stilvelman[13], Clopton[14], Allan[15]).

The question now arises whether his long illness at the age of ten was not due to diaphragmatic eventration. It is idle to speculate now, but it is very likely, in view of the protean manifestations of this disease and its periods of complete freedom from symptoms, that a diagnosis of tuberculous peritonitis would have been changed to diaphragmatic eventration, had the roentgen rays been used at that time.

* * *

Chronic Nephritis Without Hypertension, Cardiac Hypertrophy or Retinal Changes

History. Miss R. Z., aged 23, single, came to my office May 11, 1929. She was employed in a candy factory up to two years

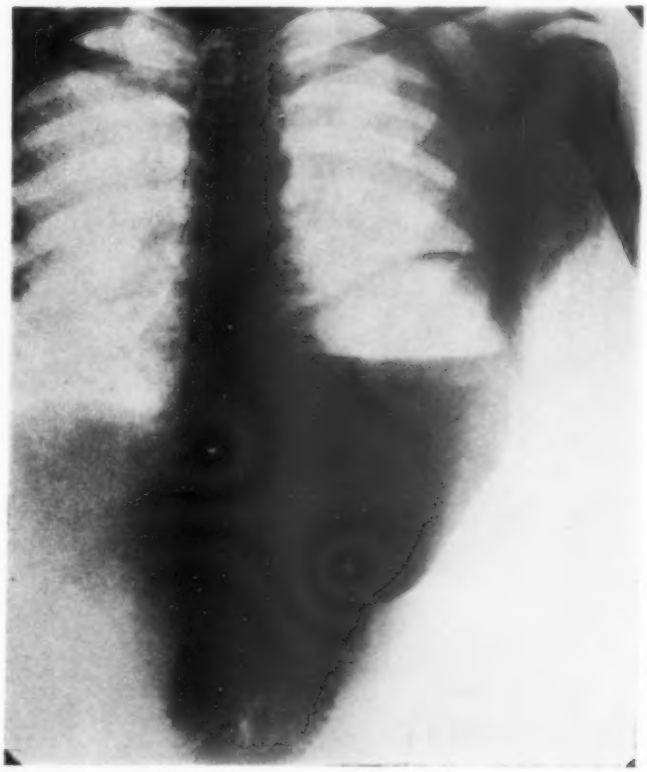

Fig. 2. Case 1. Esophagus, stomach and duodenum. Note position of cardia and large air bubble.

ago; since then she has assisted her mother in her household duties. Menstruation began at the age of sixteen and occurred every twenty-eight days, lasting three days. Habits normal. She had had the usual diseases of childhood including diphtheria and scarlet fever, for the latter of which she was kept in a hospital for contagious diseases for three months.

The patient, as well as her mother, denied that she had ever had kidney disease or that she was ever edematous. Her tonsils were removed two years before, and up to that time she had had frequent attacks of sore throat. The appendix was removed five years ago. Eight months before, she experienced dull pain in the epigastrium which was not severe and did not radiate. This pain had no relation to meals and was not relieved by vomiting or by bowel movement. Occasionally, she awakened at night with pain. During the first month of illness she vomited soon after meals; even the sight of food produced vomiting. Since then, she has vomited only at rare intervals, mostly in the morning before breakfast. The color of the vomitus was that of the food ingested. She had severe heartburn which was relieved by an alkaline water. She belched a great deal and there was a sense of heaviness in epigastrium. Her appetite had been poor throughout her life. During this illness, she had lost thirteen pounds. Her bowels were quite regular up to nine months before, since which time she has taken a daily enema or cathartics. No blood or mucus was noticed in the stools. Nocturia, frequency or dysuria, cough or dyspnea on exertion had not been noted. There was an occasional slight headache, no dizziness or

FIG. 3. Case 1. Colon and stomach filled simultaneously.

visual disturbances. She slept quite well but for the epigastric pain.

Examination. The pulse rate was 108; blood pressure, 110 systolic and 80 diastolic in both arms. The weight was 86½ pounds (39.3 Kg.). The patient was a thin, narrow individual, and her face was grayish-pale; the thyroid gland enlarged, no thrill felt or bruit heard over it; there was marked tremor of both hands; no exophthalmos present; Graefe and Moebius signs were absent; no hypersensitive skin zones present. Liver and spleen not palpable. The abdomen presented a scar due to appendectomy. There was marked epigastric tenderness.

A casual specimen of urine gave a slightly alkaline reaction; the specific gravity, 1010; a moderate amount of albumin; no sugar; microscopic examination, negative. Two blood-chemistry examinations were done in an interval of four days and gave the following results: Urea nitrogen, 166.6 to 192.3 mgms.; creatinine, 4.3 to 4.5; sugar, 108.1 mgms.; icteric index, 5; blood Wassermann reaction was negative. A urine concentration test was then carried out. The patient brought two specimens taken at 8 a. m. and 10 a. m., of which the specific gravity was 1009 and 1010 respectively. Two blood counts were taken in a four days' interval and showed 62 per cent to 57 per cent hemoglobin (Sahli), 3,500,000 to 3,100,000 red blood cells; 9,400 to 7,400 white blood cells. The differential count showed 54 per cent polymorphonuclear leukocytes, 27 per cent small and large lymphocytes, 2 per cent transitionals, 12 to 17 per cent eosinophiles. The red blood cells were very pale. There was considerable poikilocytosis and anisocytosis and an occasional microcyte was seen.

In view of the persistence of eosinophilia, the stool was examined for ova. We observed structures which suggested to us the ova of *Uncinaria americana*. In order to check up on the diagnosis of a disease with which we were not familiar, we had arranged to send the stool to a competent parasitologist who had a great deal of experience in tropical diseases. Unfortunately, the patient's relatives refused to cooperate. Perhaps it ought to be mentioned that this patient had never visited a tropical country. The benzidine test was negative. There was free hydrochloric acid present in the gastric contents.

The eye grounds were examined by an ophthalmologist who found "both fundi entirely negative".

Radiographic examination revealed no abnormalities in heart and lungs. A barium test meal was then administered. There was no evidence of organic disease in stomach or intestines. There was considerable gastroptosis, stomach reaching 12 cm. below the crest of the ileum; duodenal stasis was present to a considerable degree. After five hours both stomach and duodenum were empty.

The patient died eleven months later.

Comment

This patient had been treated for six months for gastric ulcer by an eminent gastroenterologist, thoroughly acquainted with his specialty and on the teaching staff of a large university. Nevertheless, a blood urea done by a competent technician would have done more to point towards a proper diagnosis than all the experience and learning of this clinician. The presence of albumin in the urine does not have the same significance as the high urea in the blood. Had this patient presented all the classical symptoms of chronic nephritis, the clinical diagnosis would have been evident and laboratory methods might have been dispensed with or only used to corroborate it. However, this was an atypical case (Bannick[16], Bennett[17], Fishberg[18]), and offered little, if any, clinical evidence of the true condition. Here, the clinical laboratory, impersonal and objective, made the diagnosis.

* * *

Unusual Case of Cholelithiasis

History. Mrs. S. L., aged 55, born in Russia, came to my office, Jan. 10, 1929. She

had been married 36 years and had never been pregnant. The menopause set in ten years before. When in Russia, she had the following diseases: pneumonia at the age of 18, malaria at 25, cholera at 29 and typhoid fever at 36. She had had two minor operations on the uterus per vaginam, one at 27, and the other when 30 years of age. Their exact nature could not be determined. At 40 she was operated upon for an ischiorectal abscess; at 46 a left intraligamentous cyst was removed. One year before, she had an attack of severe cramps and pressure in epigastrium, which radiated to the back, was worse after meals, but was relieved by bowel movements. The pain was so severe at times that it kept her awake at night. Within the last month she had lost nine pounds, and in the preceding eleven months, twenty-one pounds. She was never jaundiced.

Examination. There was definite and constant tenderness and rigidity in the right upper quadrant of the abdomen. Rocking the liver caused much pain. The history and physical findings were so typical of cholelithiasis that this diagnosis was made on clinical grounds by Dr. Joseph Rivkin who first saw this patient. I readily concurred in the diagnosis.

Examinations of urine, feces, gastric contents and blood were negative.

A barium test meal was administered. Radiographic examination of gastro-intestinal tract revealed a normal stomach; duodenal cap was indented at its outer border by an oval mass and a dense tube projected from the lower part of this mass (figure 4). After five hours, the stomach was almost empty, but the oval mass with the tube remained unchanged and continued so on the twenty-four, forty-eight and seventy-two hour plates.

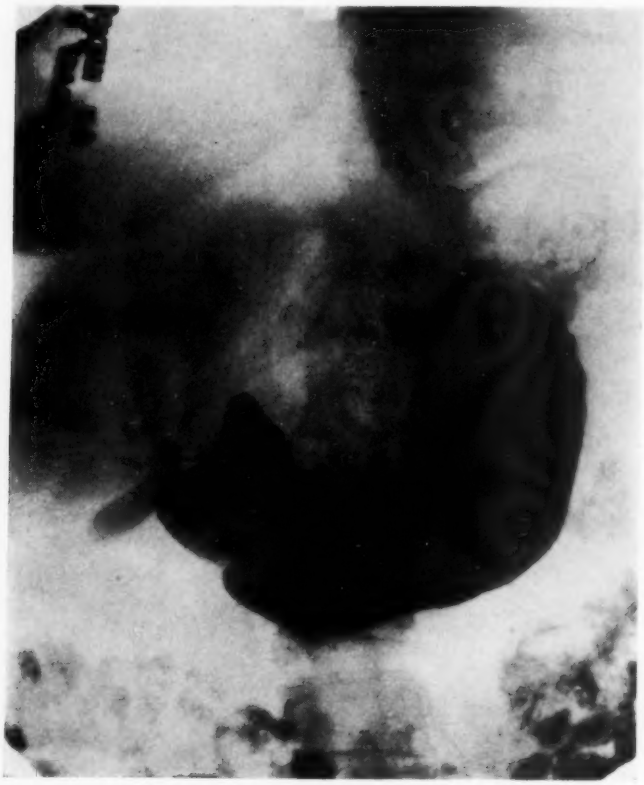

FIG. 4. Case 3. Duodenal cap is indented at its outer border by an oval mass. A dense tubular-appearing shadow projects from the lower part of this mass.

Cholecystograms were taken sixteen hours after administration of tetra-iodophenolphthalein by mouth. The gall bladder was not visualized and only the structures described above were seen. Roentgenograms were then taken twelve days, four weeks and six weeks later. The findings remained unchanged up to the six weeks' plate when a slight change was noticed (figures 5 and 6).

Interpretation of the plates was difficult. It was thought that a duodenal diverticulum was present. However, since the symptoms continued, patient submitted to an operation which was performed by Dr. John Linder at the Brownsville and East New York Hospital. The operative findings were as follows: The dense, tubular structure represented the lower part of the gall bladder which was tightly filled by a lime cast. The oval mass above it represented the upper part of the gallbladder and was tightly filled by a large cholesterin stone.

Comment

In this case the clinical diagnosis was correct while the roentgenograms only served to confuse us. This much must be admitted. The real fault, however, did not lie with the method but with its incorrect application. Had I taken a plain plate of the abdomen *before* administration of barium a duodenal diverticulum scarcely could have been suspected, and the diagnosis of gallstones would have suggested itself. Renal stones could have been ex-

Fig. 5. Case 3. After twelve days, the oval mass and dense tubular shadow remain unchanged.

cluded, if necessary, by pyelograms. Furthermore, had this precaution been taken, the exact size and shape of the stones could have been predicted accurately, whereas, clinically, this was impossible. It is therefore clear, from this case, that in the radiographic examination of the abdomen, no barium is to be administered before a study is made without it.

Conclusions

Laboratory methods in clinical medicine have recently been subjected to severe criticism on the ground that they tend to impair the physician's powers of observation and his clinical sense. In defense of these methods I wish to emphasize (1) that they are an extension of the clinician's senses, a gain far outweighing any possible loss of sense acuity; (2) that the careful application of these methods is responsible for most of the progress we have made; and (3) that they are not intended to replace older clinical procedures.

The shortcomings of the laboratory are undeniable. These can be overcome only by giving further attention to their improvement, and not by reverting to the sole use of the unaided

Fig. 6. Case 3. After six weeks, the dense tube is surrounded by a lighter oval area.

senses and to such vagaries as "personality" and "intuition".

My conclusions are illustrated by the following cases:

1. Diaphragmatic eventration. This patient had been diagnosed as a neurotic. His history and behavior strongly suggested this, but the roentgen rays revealed his true condition.

2. Chronic nephritis without hypertension, cardiac hypertrophy, or retinal changes. This was an atypical case in which all the cardinal clinical symptoms of chronic nephritis were absent. On the contrary, the symptoms were so strongly indicative of gastric ulcer that for six months this patient was treated for this condition by a competent gastroenterologist. Without the chemical study of the blood, the diagnosis was almost impossible.

3. Unusual case of cholelithiasis. This was correctly diagnosed by the history and physical examination, but was confused by the improper use of the roentgen rays. Had a plain plate of the abdomen been taken before administration of the barium, this error might have been avoided. This case, then, illustrates the value of the older clinical methods as well as the importance of using the newer methods properly.

REFERENCES

[1]Darwin, Charles: The descent of man and selection in relation to sex, ed. 2, New York, D. Appleton & Co., 1899, p. 34.

[2]Ibid, p. 32.

[3]Assman, Herbert: Die klinische Röntgendiagnostik der inneren Erkrankungen, ed. 4, Leipzig, F. C. W. Vogel, 1928, p. 438.

[4]Hirsch, Carl: Zur klinischen Diagnose der Zwerchfellhernie, München. med. Wchnschr., 1900, xlvii, 996-999.

[5]Leichtenstern: Zur Diagnose der Hernia diaphragmatica, Berl. klin. Wchnschr., 1874, xi, 497-500; 515-517; 539-542.

[6]Carman, Russel D., and Fineman, Solomon: The roentgenologic diagnosis of diaphragmatic hernia with a report of seventeen cases, Radiology, 1924, iii, 26-45.

[7]Soresi, Angelo L.: Diaphragmatic hernia: its unsuspected frequency: its diagnosis: technic for radical cure, Ann. Surg., 1919, lxix, 254-270.

[8]Funk, Elmer H.: The diaphragm; anatomic and physiologic considerations; methods of examination; conditions associated with altered positions of the diaphragm; diaphragmitis, Med. Clin. N. Am., 1921, iv, 1045-1082.

[9]Funk, Elmer H., and Manges, W. F.: Eventration of the diaphragm with report of a case, Am. Jr. Med. Sci., 1921, clxii, 348-352.

[10]Walton, Henry J.: Eventration of the diaphragm, Am. Jr. Roentgenol., 1924, xi, 420-426.

[11]Hitzenberger, Karl: Das Zwerchfell, Wien, J. Springer, 1927, p. 152.

[12]Pepper, Perry O. H.: Diseases of the thorax and its viscera, including the heart, lungs and its blood vessels, Progr. Med., 1920, iii, 17-136.

[13]Stivelman, B. P.: False pneumothorax, Jr. Am. Med. Assoc., 1920, lxxiv, 12-14.

[14]Clopton, Malvern B.: Eventration of the diaphragm, Ann. Surg., 1923, lxxviii, 154-163.

[15]Allan, W.: A case of eventration of the diaphragm, Virginia Med. Month., 1920-1, xlvii, 305-307.

[16]Bannick, Edwin G.: Severe chronic glomerular nephritis without hypertension, cardiac hypertrophy, or retinal changes: report of two cases, Arch. Int. Med., 1927, xxxix, 741-747.

[17]Bennett, Izod T.: Nephritis, its problems and treatment, Oxford University Press, 1929, p. 26.

[18]Fishberg, Arthur M.: Hypertension and nephritis, Philadelphia, Lea & Febiger, 1930, p. 370.

Auriculoventricular Nodal Rhythm*†

With Report of Cases

By Carl V. Vischer, M.D., F.A.C.P., and Lowell L. Lane, A.B., M.D.,
Philadelphia, Pa.

EXPERIMENT has demonstrated that not only the sinus node but the A-V node, the auriculoventricular bundle of His and the bundle branches, with their arborizations have the capacity for impulse production. This fact was established by the Stannius' ligatures. If a ligature is tied around the sinus of a frog's heart, or placed below the level at which the cardiac rhythm is generated, the heart ceases to beat temporarily below the level of this ligature, but after a brief standstill takes up a regular rhythm of its own. This new rhythm is generated in a lower center and is slower in rate than the original sinus rhythm. Likewise if a ligature is placed at the auriculoventricular border a short pause in the contractions is followed by the initiation of a new rhythm which has its origin in the ventricle (idioventricular rhythm), and is still more retarded in rate. These experiments demonstrate that the lower centers have the capacity for impulse production and that the lower the origin of the impulse in the Purkinje system the slower is the rate of impulse generation. In other words the rôle of pacemaker normally held by the sinus node may, under certain circumstances, be usurped by one of the lower centers.

The center producing the fastest rhythm always controls the heart. As long as the sinus node remains the center of most rapid impulse generation it retains the rôle of pacemaker. The lower centers may gain control over the heart under one of three conditions: in case of depression of sinus rhythmicity, interruption in conduction, or increased excitability of a lower heterotopic center. As long as the sinus node retains its normal rhythm the rapidity of the impulse formation destroys the stimulus to impulse production in the lower heterotopic centers, thus preventing their functioning.

Depression of the sinoauricular node usually results in the automatic transference of the function of impulse production to the auriculoventricular node. This is the phenomenon of nodal rhythm. Experimentally such a condition may be produced by a number of different procedures. Destruction of the S-A node or obliteration of its blood supply will result in the establishment of a nodal rhythm. Cooling of the S-A node will produce the same result. Increased excitability of the

*From the Department of Medicine, Hahnemann Medical College and Hospital, Philadelphia, Pa.

†Received for publication, June 1, 1931.

A-V node, caused by a warming of this structure, precipitates a similar transference of the pacemaker. Lewis has demonstrated that nodal rhythm may also follow interference with the nerves of the heart, especially combined stimulus of the right vagus and left sympathetic nerves.

It is now generally accepted that the A-V node is responsible for certain rhythms, characterized by simultaneous contraction of auricle and ventricle. Proof that such a rhythm has its origin in the A-V node was shown by the following experiment: If, during auriculoventricular rhythm, the septum is cooled on the ventricular side of the node, or as Meakins has demonstrated if the bundle is clamped, the ventricle fails in its responses, while the auricle continues to contract. Probably the most convincing evidence that the new rhythm arises in the A-V node is that the rhythm is retarded by cooling and accelerated by heating the region of the node.

Experiments by Lewis have shown that the right vagus has a greater retarding influence upon the sinus rhythm than has the left. The influence of both vagi on rhythm production in the A-V node is powerful. The left sympathetic nerve seems to accelerate rhythm production in the A-V node markedly and to a greater extent than the right nerve, as shown by the fact that isolated stimulation of this nerve may suffice to induce an A-V rhythm.

Clinically the action of the vagus is probably responsible for the depression of the S-A node. The A-V node is then allowed to escape. An escaped beat is a contraction resulting from a stimulus from a lower center and occurs when there is a long pause in the sinus rhythm as in expiration, blocked auricular extrasystole, sino-auricular block, compensatory pause in ventricular extrasystole or partial heart block. An escaped beat differs from an extrasystole in that the former occurs late in the sinus interval, while the latter is a premature contraction. The incidence of an escaped beat is not infrequently noted, but a true nodal rhythm is relatively rare. Auriculoventricular rhythm may be considered an escape phenomenon and is actually a series of escaped beats.

A temporary nodal rhythm may occur during the vagal slowing of the heart on expiration. Atropine, through its effect on the vagus, will usually abolish an auriculoventricular rhythm, although in some cases this result is not obtained. The same drug may initiate a nodal rhythm. This effect is attributed to a primary stimulating effect on the vagus. An increased excitability of the A-V node may account for the failure of atropine to abolish nodal rhythm in some cases. It has been demonstrated both experimentally and clinically that the vagus is capable of controlling the rhythm produced in the A-V node. Stimulation of the sympathetic by exercise may abolish such a heterotopic rhythm.

Electrocardiographic tracings in instances of nodal rhythm exhibit several characteristic features. The initial and final deflections of each individual complex show the same features as are noted in the nodal type of extrasystole: first, a negative or inverted P wave due to retrograde conduction in the auricle; and second, a shortened P-R

interval. Rarely an upright P wave is encountered, which is explained either as a mechanical stimulation of the auricles by ventricular contraction if the P wave follows the QRS complex, or by a variety of intraauricular block with deviated conduction in the auricle. The ventricular complex is of the normal supraventricular type. The P wave may precede the QRS complex, become buried in it, or follow it, depending on the origin of the stimuli, whether it be in the upper, middle or lower portion of the A-V node (figure I). The last type in which the stimuli, for contraction, arise in the lower zone of the node is the one most commonly noted in the graphic tracings. In such an instance the ventricular contraction precedes the auricular beat, thus giving rise to an R-P interval in place of the normal P-R interval of sinus rhythm. A résumé of two cases observed within the past year is here presented.

Case I. A man, aged 46 years, admitted to the medical ward with the complaints of dyspnea and of cough. No information of clinical importance could be obtained from his past history. He first noticed shortness of breath two years previously. This had increased progressively, and was accompanied by a cough productive of blood-streaked, watery sputum several days previous to admission. Examination revealed a cardiac enlargement to the left, a poor cardiac muscle tone, and a mitral systolic murmur. The pulse was regular in rhythm, rate 52, and the blood pressure 210/130. There was evidence of congestion at both lung bases. A Wassermann test taken the day after admission was negative. The patient failed to respond to appropriate medication, dyspnea increased, pulmonary edema supervened, a progressive fall in the systolic blood pressure was noted, and he died three days later of cardiac failure.

It is interesting to note that the pulse rate remained between fifty and sixty until the time of death, despite the failing cardiac function. This clinical factor was suggestive of heart block. An electrocardiogram taken two days before death revealed an auriculoventricular nodal rhythm with the characteristics previously described (figure 2).

Case II. A woman, aged 43 years, had been troubled with palpitation and dyspnea, with occasional precordial pain on exertion for a period of several weeks. Three years previously she had received treatment over a period of eight months for an active luetic infection, resulting in a negative Wassermann reaction. The patient stated that she was quite well until the onset of the above features in February of the current year. Clinical examination showed evidence of cardiac enlargement to the left, by physical signs and fluoroscopy, an apical systolic murmur and an accentuated high-pitched aortic second sound. The pulse was regular, rate 50, and the blood pressure 130/80. There was no evidence of congestive heart failure. A diagnosis of cardiovascular syphilis seemed justified. Electrocardiographic tracing made at this time showed the presence of an A-V nodal rhythm (figure 3).

This patient received 1/25 grain of atropine sulphate hypodermatically to note its effect on the cardiac rhythm. A tracing taken fifteen minutes after injection showed a persistence of the nodal rhythm, with some increase in rate (figure 4). This result may be explained either as a primary stimulating effect on the vagus, or an enhanced activity of the A-V node. Six days later, with the return of sinus rhythm (figure 5), it is interesting to note that the patient experienced marked improvement in her symptoms of dyspnea and precordial distress.

The phenomenon of auriculoventricular nodal rhythm is a rather rare clinical finding. In a survey of the last 1800 tracings taken in the Electrocardiographic Department of Hahnemann Hospital only four cases were recorded. The condition is usually recurrent in character and of short duration. It is often noted in alternation with normal rhythm. In unusual cases the

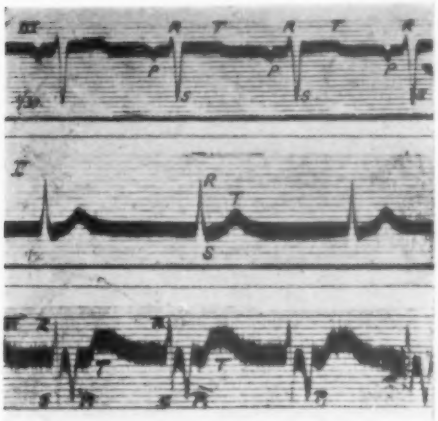

FIG. 1. Examples of three types of nodal rhythm. The upper tracing shows inverted P wave preceding the ventricular complex, resulting from impulses originating in the upper portion of the A-V node. In the type exemplified in the middle tracing the impulses arise in the middle portion of the node, causing a simultaneous contraction of auricle and ventricle. The P wave is buried in the QRS complex. Lower tracing shows most common type of A-V rhythm in which the lower portion of the node is site of impulse generation. Inverted P wave follows QRS complex.

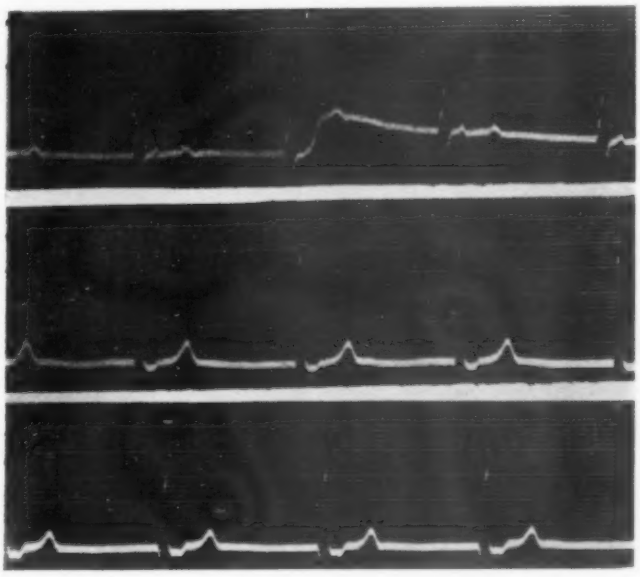

FIG. 2. Electrocardiographic tracings of case I, showing A-V rhythm of most common type, with negative P wave, preceded by the ventricular complex.

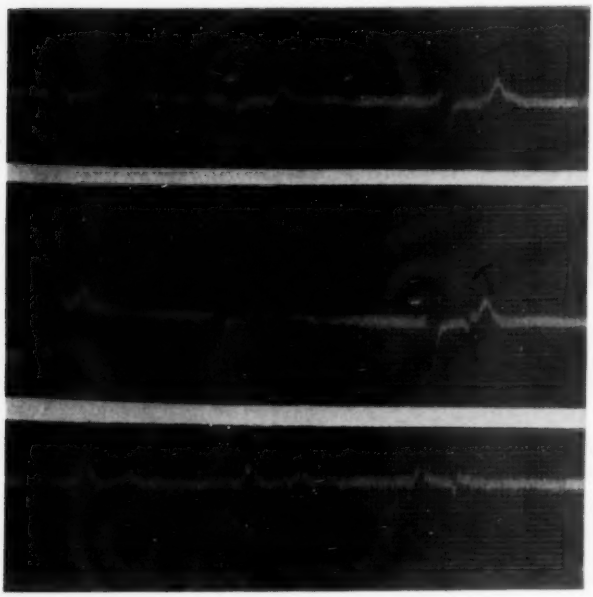

FIG. 3. Graphic record of case II. An A-V nodal rhythm resulting from impulses originating in lower portion of node.

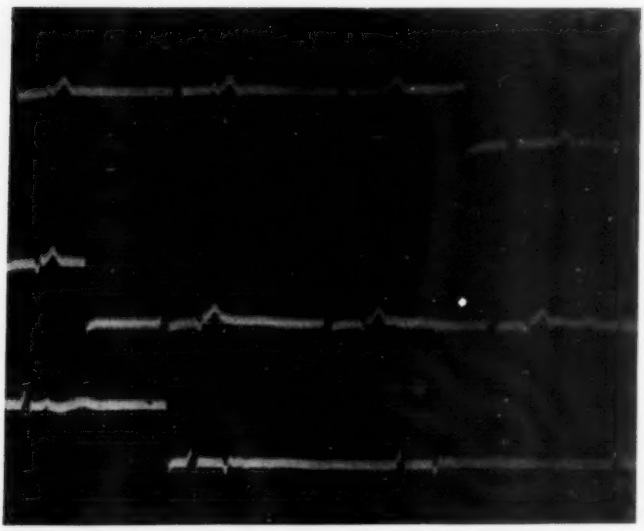

FIG. 4. Case II following administration of 1/25 of a grain of atropine sulphate. Note persistence of nodal rhythm with some acceleration in rate of impulse production.

A-V node may control the cardiac rhythm for weeks, or even months, at a time. Definite clinical evidence of its presence is usually lacking, except that in cases where the auricle and ventricle contract simultaneously, regurgitation from the right auricle may produce a large wave in the jugular vein. In such instances venous tracings will show a more or less simultaneous occurrence of the A and C waves, producing a large single wave. The heart rate is usually slow, between forty and fifty per minute in most cases, which represents the rate of impulse production in the auriculoventricular node.

Such a heterotopic rhythm may or may not be associated with cardiac disease. The two cases here reported did occur with definite evidence of cardiac dysfunction. Nodal rhythm, *per se*, is an interesting finding, but is apparently of little or no clinical significance. The importance of the condition is evidenced in the necessity of differentiating it from the vastly more serious entity of heart block suggested by the presence of bradycardia, which is common to both disturbances. A bradycardia of sinus origin must also be considered. Diagnosis must usually be made by the electrocardiogram. Prognosis and treatment in cases of auriculoventricular nodal rhythm are entirely dependent upon the associated cardiac pathology.

The authors desire to express their appreciation to Miss Florence Holstein, technician, for her cooperation in preparing the electrocardiograms used in this paper.

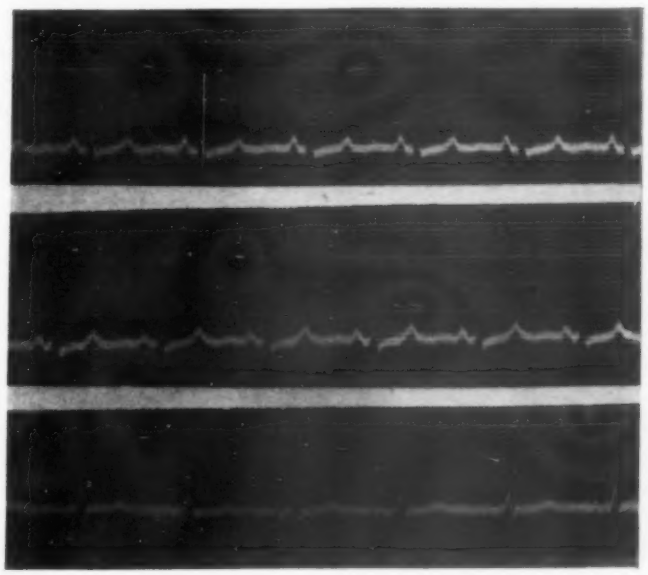

FIG. 5. Tracings of case II six days following atropine injection. Return of normal sinus rhythm.

REFERENCES

LEWIS, T.: The mechanism and graphic registration of the heart beat, 1921, Paul B. Hoeber, New York.

LEWIS, T.: Clinical electrocardiography, 1928, Shaw and Sons, Ltd., London.

EAST, C. F. T., and BAIN, C. W. C.: Recent advances in cardiology, 1931, P. Blakiston's and Sons, Philadelphia.

PARSONNET, A. E., and HYMAN, A. S.: Applied electrocardiography, 1929, Macmillan Company, New York.

WHITE, P. D.: Heart disease, 1931, Macmillan Company, New York.

GÉRAUDEL, É.: The mechanism of the heart and its anomalies, 1930, Williams and Wilkins Company, Baltimore.

Creative Evolution and Prosperity

THE theory of "creative evolution" recognizes the fact that the whole may be more than the sum of its parts. A dog is more than an aggregate of carbon, hydrogen, nitrogen, and phosphorus, supplied with certain energy; and its nature never could be discovered by a study of the elements of which it is made. A symphony orchestra is more than an aggregate of individual musicians. Only when each player finds his personal expression in a unity with the entire orchestra and in contributing to the most perfect total effect does a great symphony appear.

American prosperity, through exceptional circumstances, reached a high crest. That climax of prosperity may prove to have been but the rare crest of a wave if those in business see themselves as independent units, each striving primarily for his own satisfaction. For prosperity of a high type to be permanent it will be necessary for individual and corporate interests to be subordinate to the aim of producing a high and generally distributed well-being. Just as a football team cannot greatly succeed if it is made up of star players, each bent on distinguishing himself regardless of the game as a whole, so it is with business.

Those familiar with the motives of some "re-financings" and mergings of the recent past, with the selling of great quantities of stock to the public at high prices, as well as other business practices of the period of prosperity, are aware that the subordination of special interests to the general good is not yet a dominant trait of American business.

Permanent prosperity on a high level will evade us until that spirit is dominant. Except as economists take such factors into account, the disappearance of prosperity will continue a mystery. If creative evolution can take place in our economic life, general prosperity can increase to levels heretofore unknown. It will be better if this comes from the spirit of the times, rather than from enforced government action.—(A. E. M., in *Antioch Notes, Vol. 9, No. 7, January 15, 1932.*)

Somatic Disorders of Functional Origin*†

By S. KATZENELBOGEN, M.D., F.A.C.P., *Baltimore, Md.*

SOMATIC disorders of functional origin present quite a common problem in general medicine as well as in any of its special branches. In dealing with this subject one question immediately arises: Is it possible, or is one altogether justified in drawing a hard-and-fast line between "functional," so-called, and "organic"? Those "organicists" who believe in such a clean-cut demarcation, who are trained to think in terms of lesions exclusively, and to whom therefore complaints and even objective disorders have a meaning only when they can be reasonably explained by a detectable organic alteration, would do well to remember the following facts: In the domain of Neurology, the last three decades have witnessed an extension of the group of organic diseases of the cerebrospinal nervous system at the expense of the group of functional diseases. I allude to such diseases as chorea, athetosis, Parkinson's disease, and Thompson's disease, which have been successively transferred from the functional into the organic group. This fact may be gratifying to the organicist, but it should also serve as a warning against a loose attitude towards functional disorders, which, for many physicians, under the label of "neuropathy," are quite equivalent to "imaginary malady". I should also like to call your attention to some facts in other domains: Clinical and anatomo-pathological observations have brought to light the concept that the morbid manifestations of such an organic disease *par excellence* as angina pectoris appear to be controlled by a functional factor. Such a belief has its basis in the fact that one finds on postmortem examination sclerosis of the coronary artery in persons who never had *anginal attacks*. Moreover, in certain patients who did suffer from typical attacks of angina pectoris, and displayed evidences of a marked excitability of the vegetative system, the anatomical examination did not reveal lesions in the myocardial vessels; nor could any lesion of the vegetative system be discovered. It has also been found that the occurrence and frequency of attacks in such organic diseases as symptomatic asthma and biliary lithiasis are more or less conditioned by the functional state of the autonomic nervous system. These observations sufficiently demonstrate the fragility of the lines drawn between organic and functional. Besides, the

*Presented at the Fifteenth Annual Clinical Session of The American College of Physicians, March 25th, 1931, Baltimore, Md.

†From The Henry Phipps Psychiatric Clinic (Department of Internal Medicine) Johns Hopkins Hospital, Baltimore, Md.

usual opposition of "functional" versus "organic" does not seem to rest upon a sound basis; for the so-called functional disorders are associated with definite changes in secretion and motility of organs; they are accompanied by alteration of the physicochemical status of the blood and other bodily fluids. These modifications present organic disorders going hand in hand with the altered function, and, needless to say, are capable of causing as much distress as structural disorders. It would therefore seem more appropriate to use the term physiogenic as applied to both "organic" and "functional" disturbances, bearing in mind that in organic disorders the altered function is accompanied by histological changes and in purely physiogenic disorders, only physicochemical changes may be found. As to the "psychogenic bodily disorders" they are obviously also attended by altered physiological functions. The term psychogenic only implies the notion that certain psychological factors take part in the causation of the physical disorders. Functional hyperchlorhydria, for instance, may be induced by certain physical stimulants, highly seasoned food, alcohol, or by affective disorders, in which case it will be of psychogenic origin.

In this necessarily sketchy presentation, I should like to bring to your notice some of the commonest functional disorders. Pain is one of these troubles and it calls for special attention not only on account of its distressing, incapacitating effect, but also because this is a warning that something is wrong. Excluding from the discussion pain associated with detectable physical disease, one may ask whether purely psychic factors are able to induce pain in apparently normal individuals? In answering this question I refer to the following two cases, which, in my judgment, illustrate psychogenic pain:

1. A man, 43 years old, graduate chemist, had suffered from a severe attack of sciatica for over a year. The recovery was complete and no relapse had taken place during the six years he was known to me. He presented, however, the following curious phenomenon: On many occasions when reminded of his previous illness, or when he was thinking about the possibility of a recurrent attack, he experienced severe, but fortunately very transitory pain in his leg, without developing, otherwise, neurasthenic or hysterical features.

In the second case, suggestion of a very protracted character was evidently the cause of the evil.

A young man, a medical student, had been suffering from dull pains in the back of the head. The history revealed that the trouble started after he had been painfully impressed by the death of a classmate from cerebral tumor. Notwithstanding the fact that his fear of also having a tumor was dissipated by the entirely negative physical examination, his head sensations, more or less pronounced, had persisted during the four years I knew him.

Psychogenic pain may also be exemplified by the following two cases observed in this clinic.

A woman, among other troubles, complained of soreness of the tongue. This complaint became particularly prominent and persistent after a suspicious diagnosis of cancer, based on some enlargement of the papilla on the base of the tongue, was made. The patient continued complaining of pain long after the local symptoms had subsided.

Another patient came to this clinic because of attacks of soreness of the tongue which had taken place on several occasions during her lessons in school and for which talking was blamed. I must add that there was a certain physical background in the beginning, namely, throat and sinus troubles which may raise the question of referred

pain. But during her stay here, in spite of the fact that she still had remnants of her previous condition in the throat and antrum and was very talkative, she did not have any pain. It therefore seems plausible to assume that we are confronted here with pain of recollection analogous to the case of sciatica which I have just recorded. The fear of losing her position should attacks of pain which she originally experienced while teaching recur, made her on certain occasions think vividly of this experience during her lessons and feel accordingly.

As to the mechanism of psychogenic pain, the following conjectures may be considered: 1. Psychic stimulation with response of certain areas of the brain, such as the thalamic region, center of pain sensations, from which pain is projected to the periphery, as it is assumed to be in organic central pain. 2. Pain is known to be frequently associated with vasomotor disorders regarded by some writers, not without plausible reason, as the immediate cause of pain, be it of central or peripheral origin. The last mentioned hypothesis seems the more plausible because pain is a common complaint of psychoneurotic individuals who, besides, usually display evident vasomotor disturbances.

Another complaint not substantiated by somatic findings is fatigue. It may have but little bearing on the muscular strength or muscular tone. It will be found that this type of fatigue is rather closely related to lack of interest in the particular effort which causes it, and may therefore be termed selective fatigue. Being functional this kind of fatigue is, however, not infrequently associated with loss of weight, as a result of loss of appetite, and inadequate food intake.

Functional cardiac troubles constitute perhaps the most conspicuous chapter in functional pathology. Their symptomatology shares, with that of functional disorders of other vital systems, the common character of being mainly subjective. Sensation of pressure, pain in the cardiac region, sometimes with radiation to the left arm, palpitations, inability to take a deep breath are common complaints. The latter, usually of a rich variety are, however, not referred to the heart only. The objective examination will reveal symptoms with which we are all familiar, such as tachycardia of the transitory type, hyperkinesis, extrasystoles, systolic murmurs usually localized in the mesosternum, and vasomotor lability. Here I should like to call attention to a special type of cardiac neurosis. That is the syndrome known in the literature of war neurosis as "irritable heart of the soldier", or more commonly in the general medical literature as "effort syndrome", or "neurocirculatory asthenia". As the last named term suggests, the troubles are referred to both the circulatory and nervous systems.

In addition to complaints and objective symptoms common in functional heart disorders, there are other features which dominate the clinical picture: Fatigue, usually more pronounced in the earlier part of the day, dizziness, giddiness particularly liable to occur when the sitting position is changed to a standing one; general shakiness, fainting, with or without complete loss of consciousness; excessive susceptibility to cold, sleeplessness, inability to fix the attention, and lack of interest. This syndrome may be found in juvenile cases, and in adults, and is not infrequently responsible for the accusation of "laziness" among school boys

and girls. In many cases a constitutional origin is suggestive. In others, this syndrome appears to be a sequel of a toxic-infectious condition. In the last named cases one will be confronted not infrequently with the dilemma as to whether the protracted effect of the toxic-infectious condition is solely to be blamed, or whether the oversolicitous family or doctor are not partly responsible for keeping the patient away from his duties longer than is absolutely necessary, with the consequent worries about waste of time and loss of the patient's selfconfidence.

Finally, I want to call attention to some of the functional gastrointestinal disorders. Numerous experimental investigations and clinical observations of patients with gastric fistula, observations having the value of experiments, are available, which point to certain etiologic factors of the socalled nervous indigestion. These studies demonstrate that affective imbalance, and mainly acute emotion (anger, anxiety, fear, etc.) strongly influence the gastric secretion. The latter usually increases under pleasant emotions (sight or smell of food, pleasant environment while eating) and decreases under unpleasant ones. This relationship between secretions of the digestive system, and emotions is an old notion familiar even to laymen, as indicated by the ancient test used in India to pick out a thief from a group of suspicious persons, a test in which each person is given a bowl of rice and the criminal is detected by his dry mouth, and inability to insalivate the rice.

The motor function of the alimentary canal may be equally controlled by psychic factors. Here again I refer to the common observation of physiologists, demonstrating that uneasiness, discomfort, and anger, experienced by animals, immediately induce cessation of the gastrointestinal movements. If under these conditions the splanchnic nerves are cut, the inhibition will be reduced to a great extent. On the other hand, clinical and radiological investigations on men, also demonstrate the inhibitory or stimulating effect of emotion on the gastrointestinal motility. The barium meal may remain in the stomach for many hours without moving, in individuals under nervous strain in whom no organic alteration is recognizable. Psychic stimulation may also have the opposite effect, namely, to increase the motility. In this respect the following case may be illustrative:

A man who, in his capacity of a high official in an international institution, had to attend banquets and various social gatherings, suffered from the following trouble: On many of these occasions, as soon as he started eating he had to leave the table, summoned by the call of nature. No organic cause for this trouble could be detected. On the contrary, the history of the disease was very illuminating. This man had been brought from obscurity into prominence during a revolution in his country. From a modest social standing, an humble member of the socialistic party, he suddenly found himself a representative of his country in the institution of highly trained diplomats. This turning point which threw him into a *milieu* and conditions not usual to him, was the beginning of his troubles, obviously on an emotional basis.

Finally, radiological examinations showing the reverse current in the digestive tract in certain patients, while under emotional strain, substantiate the clinical observations of vomiting in neurotic individuals, in whom no causes other than effective disturbances can be revealed.

The problem of etiology, pathogenesis of functional disorders in general, and of the gastrointestinal disorders in particular has given rise to theories implicating the autonomic nervous system. These theories advocating the conception of sympatheticotonia and vagotonia, that is, of an imbalance between the two antagonistic parts of the visceral nervous system, had a wide vogue after they were propounded by Eppinger and Hess in 1910. Now, we should all be familiar with the fact that the subdivision of neurotic individuals into two clean-cut reaction types is not warranted by clinical observation. It is true that in certain subjects there are evidences suggestive of a certain predominance of one set of the vegetative nerves—the sympathetic or the parasympathetic. But what we commonly meet with in neurotic individuals is a disturbed function involving both portions of the visceral nervous system. One usually observes a dystony which may manifest itself not only by a relative increase of the controlling power of one portion, but also by an increase or a lowering of the tone of the whole visceral nervous apparatus.

From the etiologic standpoint the malfunction of the autonomic nervous system raises the question of the rôle of constitution, the rôle of toxic-infectious conditions and other physical factors. Another element weighing heavily in disorders of the viscera under the control of the vegetative system, an element which is commonly not given adequate consideration by the physician at large, by the internist, and general practitioner, is the emotional one. Of course, the relationship of cause and effect is open to debate. One may ask whether the excessive responsiveness of the visceral organs to certain life situations is conditioned by inherent or acquired malfunction of the autonomic nervous system or of the viscera themselves under its control, or are these excessive reactions only a proportionate response to strong emotion proper to the psychobiological endowment of the individual involved? Leaving this question out of the discussion it suffices for our purpose to note the fact that affective imbalance, arising from life conflicts and not infrequently from pleasant life experiences also, is attended by more or less pronounced reactions of the vegetative organs. Moreover, it is well to bear in mind the notion ably advocated by Cannon, that affective disorders, when they repeat themselves, are able to sensitize the vegetative system which will then react excessively, even to a mild emotional upset.

In concluding I would like to emphasize the three following points:

1. The socalled functional disorders are not "imaginary maladies," and they may cause as much discomfort and distress as organic structural diseases.

2. Functional disturbances may be induced by various factors, physical as well as psychological.

3. A thorough physical examination should therefore be supplemented by a no less thorough inquiry into the conditions within and without under which the troubles originated and developed. Such an investigation is obviously imperative whenever no somatic basis for bodily disorders can be found. Moreover, one should be attentive to the fact that manifestations of organic-structural diseases may also be greatly influenced by psychological factors.

Advanced Pulmonary Tuberculosis*

By Lewis J. Moorman, M.D., F.A.C.P.,† *Oklahoma City, Oklahoma*

FOR many decades the dominant theme of practically all discussions dealing with clinical tuberculosis has been early diagnosis and early treatment. While the significance of this theme is perfectly obvious and the difficulty of early diagnosis universally admitted, there still seems to be a general lack of interest in tuberculosis. This may be accounted for in part by the fact that, until recently, we have been unable to offer any constructive variation in a program which often proved inadequate. It is not surprising that many members of the profession not particularly intrigued by the interesting game of physical diagnosis, and not committed to the rather difficult task of phthisiotherapy, should manifest a certain amount of indifference.

Without intensive study there can be little first hand knowledge of the fascinating course of this versatile disease with its ever changing interplay of virulence and resistance; there can be no adequate appreciation of the fact that the relative dominance of these two factors, determines the clinical course and the pathological variants which range from those manifested through the racing catastrophe of florid phthisis to the relative security of insidious fibrosis. Since tuberculosis continues to be one of the prime factors in morbidity and is responsible for one-seventh of the world's mortality, it deserves the serious interest of the medical profession.

While it is necessary to maintain our emphasis upon early diagnosis and early treatment, it is most gratifying to be able to recount the fact that recent advances in the treatment of pulmonary tuberculosis offer a new hope to those suffering from the more advanced conditions which might otherwise be considered hopeless. Considering the three cases which Dr. Pincoffs has so kindly provided for this clinic, I am indeed pleased to say that our present therapeutic measures are sufficiently flexible to meet many of the wide variations in pulmonary pathology. Many of these variations are exhibited in the cases now to be presented and in addition to the established routine, rest, dietetic and hygienic management, they call for a discussion of the following therapeutic methods: artificial pneumothorax, the cauterization of pleural adhesions, the various operations on the phrenic nerve, and thoracoplasty.

*Clinic presented at the University of Maryland, School of Medicine, University Hospital, in the Fifteenth Annual Clinical Session of The American College of Physicians.

†Professor of Clinical Medicine, University of Oklahoma.

Case I

The first case, C.B., is a young colored woman thirty-three years of age, married, no children, but in contact with two children in the home where she is employed to do general housework. Family history negative, except contact with a luetic husband. No history of known contact with tuberculosis. Past history otherwise negative except the loss of about sixty pounds during the past year.

Present Illness. In the early part of December, 1930, while at her usual work, she developed a headache, soreness in the chest and weakness. These symptoms were promptly followed by a chill and high fever. A cough and hoarseness developed. After two days she returned to work. Her cough continued and a few weeks later it became productive with a gradual increase in sputum. Three weeks ago, March 3, she had to quit work on account of increasing weakness. She was examined in the outpatient department one week ago and was admitted to the Hospital with a diagnosis of bronchopneumonia, possibly tuberculous in character. Since admission she has shown a temperature range of 99 to 102, pulse 80 to 105, respiration 20 to 30. She has continued to cough and the record indicates that she has raised four ounces of sputum daily.

Physical examination shows limited respiratory excursion on the right, palpation is negative, percussion elicits dullness from the third rib anteriorly to the base. This dullness extends well into the axilla but there is very little impairment posteriorly. The left shows no demonstrable impairment of resonance. Auscultation reveals numerous fine and medium râles over the lower half of the right chest with large bubbling and occasional musical râles on a level with the fourth right interspace anteriorly. These râles are suggestive of the presence of cavity, though there are no other signs to aid in the diagnosis of cavity. With the exception of a few crackling râles along the left border of the heart, auscultation on the left is negative.

An examination of the sputum showed many tubercle bacilli; the blood and urine were reported negative. Blood Wassermann test was found to be four plus. A flat film of the chest made upon admission shows marked opacity in the lower half of the right lung, suggesting widespread infiltration of varying density. Extending from the lower angle of the right hilum there is an oval density with a rarified center, suggesting a cavity. Stereoscopic films made yesterday present unmistakable evidence of a cavity about five by eight centimeters in diameter. In the parenchyma of the right lung opposite the fourth rib, there is a small calcified node. This, associated with calcification about the hilum, suggests a childhood infection. The left hilum is heavier than normal and there is a generalized fibrosis with a little patchy infiltration in the lower half of the lung. (Figure 1.)

The diagnosis in this case is pulmonary tuberculosis with rapid caseation and liquefaction with cavity formation. There is evidently an acute bronchogenic spread into the lower right, giving rise to a tuberculous bronchopneumonia. The defensive reaction seems to be wholly inadequate, there is practically no evidence of fibrosis and the course of the disease would suggest that not only has there been a failure on the part of the defensive forces to react with the formation of fibrous tissue, but even the reticulum fibers have gone down under the virulent onslaught allowing the disease to progress rapidly.

However, the patient has shown a progressive gain in general physical well being since admission to the hospital which is encouraging. In spite of the apparently acute course of this case the prognosis is not utterly hopeless. The right diaphragm exhibits a fairly free excursion and it is possible that the pleural space is free. If collapse can be obtained by means of artificial pneumothorax the cavity may

be closed and the progress of the disease arrested.

I should advise this course regardless of the slight present trouble on the left side. If the overwhelming load of toxemia can be even partially lifted by pneumothorax on the right we may expect recession rather than progression of the disease in the contralateral lung. If adhesions should be encountered and if they should happen to be of such a character as to justify the Jacobaeus operation (thoracoscopic visualization and cauterization) the handicap of limited collapse might be overcome. If this should prove impractical, phrenicectomy might be indicated. If one or all of these methods should result in marked improvement and yet prove insufficient to close the cavity, cautious employment of thoracoplasty might be considered. If the contralateral lung should happen to take on a progressive phase under such procedures, partial collapse of the left lung by means of artificial pneumothorax might be undertaken, even though it should necessitate simultaneous bilateral pneumothorax. Such a case is not to be considered favorable for any type of treatment, yet the hopelessness of the case if untreated seems to warrant a trial, especially as the results in such cases are occasionally surprisingly good. The presence of an active luetic infection may exert an unfavorable influence through lowered resistance. Antiluetic treatment should be guarded because of the acute character of the tuberculosis.

Case II

The next case, A.W., is a man forty-two years of age. Family and personal history uneventful except contact with father who coughed and expectorated over a period of

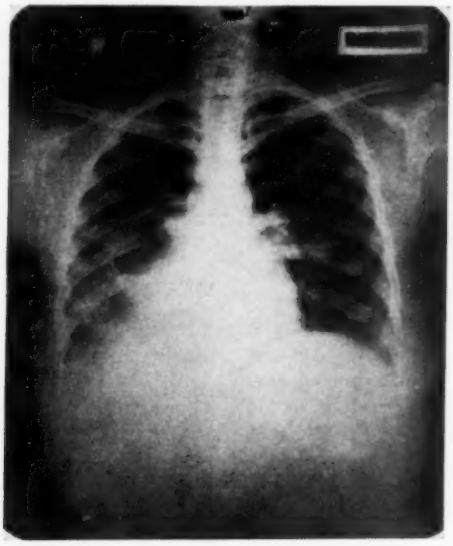

Fig. 1. Case I. Advanced pulmonary tuberculosis predominating at the right base, large cavity at the right border of the heart extending between third and fifth ribs.

many years. While there may be some question as to the diagnosis in the father's case, this cough was attributed to tuberculosis. The hospital record indicates that the onset of this patient's present illness was three months ago, when he became too weak to continue his daily routine. He developed a cough with moderate expectoration, dyspnea and fever. A close investigation with reference to his past reveals the fact that he gives a history of what might be termed a frank pleurisy two years ago and that he has coughed and expectorated for years. As may be seen later, this additional history is in keeping with the physical and pathological findings.

The patient was admitted to the City Hospital two weeks ago. Since this time his temperature has ranged from 98 to 100; his pulse from 72 to 90; respiration from 20 to 30; sputum, four ounces. He has manifested symptomatic improvement with some reduction in sputum. The laboratory reports show that the blood and urine were negative. Sputum showed many tubercle bacilli. Physical examination at the present time reveals a well developed man showing no particular evidence of loss of weight. Inspection with reference to the thorax elicits nothing of importance. Palpation is likewise negative except for moderate increase in palpable fremitus at the left apex. Percussion reveals dullness on the right from the fifth rib up, on the left from the first rib up with slight impairment extending as low as the fourth rib. Posteriorly, resonance is impaired on the right from the apex to the lower angle of the scapula, and on the left as low as the fifth vertebra.

The right diaphragm is practically immobile. Auscultation shows that the breath sounds are slightly diminished over the whole right, but exaggerated at the left apex. Fine and medium crackling râles are to be heard over the whole of the right and the upper half of the left. At the left apex coarse bubbling and musical râles are heard. These signs with exaggerated breath sounds are sufficient to warrant a diagnosis of cavity at the left apex. There are no demonstrable signs of cavity on the right side. A flat film of the chest presents unmistakable evidence of old fibrocaseous disease at both apices with definite cavity formation at the left. (Figure 2.) Above the level

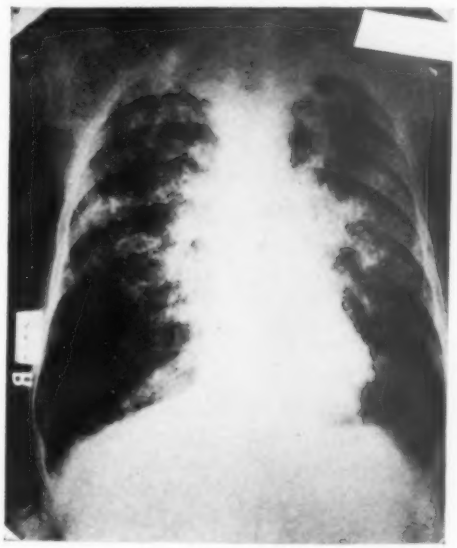

FIG. 2. Case II. Advanced bilateral pulmonary tuberculosis with cavity formation.

of the first rib on the left, the film suggests an old pleuritis with marked thickening, giving rise to the so-called pleural cap. On the right side there is a generalized patchy infiltration extending as low as the fifth rib anteriorly. There is also a similar area on the left extending from the hilum and fusing with the old pathology at the left apex.

This widespread dissemination of what appears to be recent disease is in all probability a bronchogenic spread of an old infection and we may be justified in assuming that it is contemporary with the recent exacerbation of symptoms. On the right side opposite the first rib there is an illy defined thin-walled cavity about 2½ centimeters in diameter. This evidently is likewise of recent origin. The right costophrenic angle is poorly defined and there is obvious peaking of the right diaphragmatic dome suggesting adhesions, which may be dated back to the pleurisy two years ago and which may be utilized to explain marked limitation of diaphragmatic excursion on the right as shown by percussion. You readily appreciate the fact that we have little to offer in this case, yet the low grade toxemia in the presence of such widespread disease suggests a fighting chance. Leaving out of consideration the economic phase of such a case and assuming the possibility of long continued institutional treatment, the following procedure is suggested: Since the acute manifestations are more extensive on the right with a small cavity, the walls of which are not sufficiently fibrosed to materially resist collapse, I would suggest artificial pneumothorax even though adhesions are certainly present. Partial collapse often accomplishes great good and places the patient in a condition to withstand more radical procedures. In case pneumothorax is impossible on account of obliterating pleurisy or in case basal adhesions are extensive and cannot be cauterized, the pneumothorax may be supplemented by phrenicectomy. If the results of treatment on the right are promising, circumscribed thoracoplasty might be employed to close the old cavities at the left apex.

Case III

Wm. Y., age sixty, is married and a cigar maker. Past history is negative until five years ago from which time he dates his present illness. Family history is negative. Five years ago he first noticed symptoms of nasal obstruction which was associated with cough. A few months later he had an operation for the relief of this nasal obstruction and something was removed from his nose. His cough continued with increasing expectoration. Three years ago he suddenly developed an attack of asthma which required a hypodermic before he got relief. Since that time he has had wheezing with irregular periods when he would have frank asthmatic attacks. He was admitted to the City Hospital two months ago. His temperature occasionally reached 100, but it has been normal the past six weeks. Pulse, 100 to 120; respiration, 20 to 25; blood pressure, 112/80. Sputum was sixteen ounces daily at first, now about eight ounces. Cough is worse during the night and is paroxysmal in character.

Examination reveals the classical signs of chronic bronchitis with emphysema. Wheezing and crackling râles throughout both lungs. The examination is otherwise negative except at the left apex where are to be found all the signs of fibrocaseous tuberculosis with cavity formation. The trachea is displaced to the right. The reported laboratory findings, including a blood Wassermann test, are negative. Five sputum examinations failed to show tubercle bacilli. With such large quantities of sputum one would naturally expect to find tubercle bacilli if tuberculosis constitutes the chief source of the sputum. In this case there is a his-

tory of asthmatic attacks with long continued bronchitis. It is reasonable to presume that even though the diagnosis of chronic pulmonary tuberculosis is justified there must be some associated condition to account for much of the sputum. Naturally one would suspect a bronchiectasis.

A flat film of the chest as you may see, reveals typical evidence of chronic fibroid tuberculosis at the apex of the left. (Figure 3.) Strange to say the mediastinum is displaced toward the right, which accounts for the visible and palpable displacement of the trachea toward the right. The lung fields show a generalized fibrosis with rather marked fibrosis extending from the right hilum to the right apex. This latter probably represents an old healed lesion with scar tissue and adhesions which displaced the trachea and heart toward the right, and fixed them in this abnormal position before the fibrotic pull was exerted on the left. There is evidence of diaphragmatic adhesions on both sides and perhaps a slight suggestion of bronchiectasis at the right base.

My impression in this case is as follows: an old healed tuberculous lesion, upper right; chronic caseofibrous tuberculosis with cavity formation at the apex of the left; chronic bronchitis with bronchiectasis and emphysema. The nose and throat examination revealed a pansinusitis. In this case the sputum should be examined repeatedly, concentration methods being employed. If persistently negative, cultures should be made or animal inoculation employed. The introduction of lipiodol might determine whether or not he has dilated bronchial tubes and whether the dilatations are unilateral or bilateral. If the bronchiectasis is right unilateral as suspected, or if it predominates on the right side, the right phrenic nerve might be taken. Whether or not anything should be done with reference to the upper left will depend upon the progress of the case. The patient's age and cardiac condition would render any major surgical procedure hazardous. Obviously the sinusitis should have appropriate treatment.

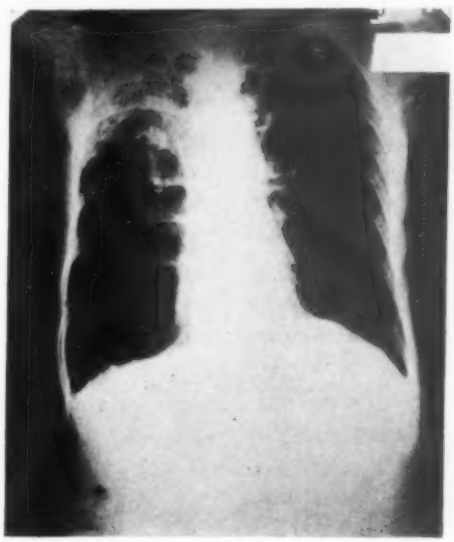

FIG. 3. Case III. Chronic fibroid tuberculosis of the left apex with a possibility of bronchiectasis at the right base.

The Diagnosis of Hyperthyroidism*†

By Maynard E. Holmes, M.D., F.A.C.P., *Syracuse, N. Y.*

THE term hyperthyroidism has been adopted in recent years to designate a symptom complex caused by hyperactivity of the thyroid gland. In the past, patients who are now classified as having hyperthyroidism together with a large group with a similar symptomatology but not of thyroid origin, were diagnosed as having Graves' disease. Kessel[1] has called attention to the latter group and applied to it the term autonomic imbalance, to designate it as a disturbance of the autonomic nervous system.

Moschcowitz[2] is of the opinion that Graves' disease evolves from a basic neuropathic personality to its full-blown clinical form when hyperthyroidism has secondarily developed. Others have described the Graves' constitution, Basedowoid, pre-Basedow and *formes frustes* as precursors of true Graves' disease. Admittedly only a very small percentage of the large neurogenic group ever develop hyperthyroidism, and prior to the development of an elevated metabolism proof is lacking that the symptom complex is in any way related to the thyroid gland, hence for this group some such terms as autonomic imbalance or neuro-circulatory asthenia more clearly implicates its neuropathic origin. The real problem in the management of these two groups of patients is not so much that an occasional neurotic individual may develop hyperthyroidism, but from a practical standpoint it is far more important to separate the true thyroid patient from the neuropathic one because they demand radically different treatment. It is perhaps unfortunate that the term Graves' disease has not been restricted to those cases having hyperthyroidism, because the two terms are commonly looked upon as synonymous. The confusion brought about by linking the neurogenic group with the thyroid gland not only leads to unnecessary thyroid surgery, but has in the past, been the cause of considerable dissatisfaction with the treatment of hyperthyroidism, and in like manner no doubt explains the disagreement which surrounds the pathology of thyroid disease.

Until the basal metabolism test was introduced as a clinical procedure in 1920, there was no dependable laboratory method to guide one in the diagnosis of thyroid disease. At present, with the help of this valuable clinical test, it would seem that there should be no valid reason for mistaking a neurosis for hyperthyroidism, and yet the error is still commonly made. Ham-

*From the Department of Metabolism, University Hospital, Syracuse University College of Medicine.

†Received for publication, June 16, 1931.

ilton and Lahey[3] report that one-third of the patients referred to their thyroid clinic, after careful study fail to show any disorder attributable to the thyroid gland.

Failure to differentiate between the true thyroid syndrome and that of neurogenic origin is in great part due to the fact that many practitioners and particularly surgeons seems to underestimate the part played by functional disorders of the nervous system as a cause of many varied symptom complexes. The surgeon, no doubt because of his daily precise technical problems, develops a frame of mind which does not sufficiently evaluate the part functional disorders may play as a great mimic of organic disease. Medical students formerly were so cautioned against making other than an organic diagnosis that later in practice, they lack the courage to do so. The present day student on the other hand, has fortunately a much more rational understanding of the importance of functional disorders of the nervous system as a great cause of ill health.

The following case reports are given to illustrate the manner in which a simple neurosis is frequently confused with hyperthyroidism:

Case I. A woman, aged 42, who had always been nervous and in poor health, complained of vague stomach discomfort and fatigue for five years. Anorexia, constipation, pain in the bowels, headache, lame back, sensitiveness to cold, insomnia and palpitation had been present for two years. She had had two Caesarean operations and her appendix had been removed for chronic appendicitis. She had been told by a surgeon that her symptoms were due to thyroid disease, and thyroidectomy urged. To confirm this opinion she consulted an internist who found a normal pulse, no enlargement of the thyroid and no evidence of thyroid toxicity. The basal metabolic rate was found to be +8. She had always worked hard, and there had always been considerable domestic and financial worries which no doubt were factors producing her symptoms.

Case II. A woman, aged 29, was admitted to the hospital with a diagnosis of hyperthyroidism with surgery recommended. Some enlargement of the thyroid had been present since 14 years of age. Nervousness, fatigue, attacks of nausea and vomiting, headache, backache, anorexia, sensitiveness to cold, palpitation, tremor and emotionalism had been present for eight years, or since marriage. There was a marked nervous tendency in her family; one brother having mental disease. The basal metabolic rate was found to be +1. Since marriage there had been considerable family and financial worries. A neurogenic cause for her symptoms seemed quite evident. Several months later after appropriate treatment for her neurosis, she was found to be almost completely relieved of her symptoms.

Case III. A woman, aged 34, who was always of a nervous temperament, developed, after considerable marital trouble, increase in nervousness, palpitation, hysterical seizures and fits of depression. There was no loss of weight, thyroid enlargement or sensitiveness to heat. The metabolic rate was —7. A surgeon was inclined to a diagnosis of hyperthyroidism but finally bowed to the opinion of an internist and neurologist that the patient was suffering from a psychoneurosis. The patient seemed obsessed with the idea that her thyroid must be removed and consulted a surgeon in another city. On the basis of a positive Goetsch test, he performed a thyroidectomy. As might be expected from the normal basal metabolism there was no relief of symptoms and within a year the patient was in an institution for treatment of her mental state.

The value of the basal metabolic test in differentiating the thyroid from the nonthyroid patient is often vitiated because of errors of technic or interpretation. When the test was introduced,

Benedict[4], DuBois[5] and others cautioned that the procedure could be of little or no value unless performed by one familiar with all the details involved, and using only the most accurate methods. Certain manufacturers have simplified the apparatus and the compilation of the results to such a degree that it is very easy for considerable error to occur. While there can be little doubt that a well trained technician can accurately perform this valuable clinical test, it is quite another thing to have it performed as it commonly is by one who has had no training except perhaps from a circular or a salesman. The performance of this test which carries so much weight in diagnosis should be as carefully guarded as, for example, the Wassermann test.

Perhaps the greatest error in the interpretation of the results of the basal metabolic test is the significance attached to figures reported only slightly above the limits of normal. It is a common observation to obtain at an initial test figures of from +15 to +35 which at a second or third rate-taking will be found well within the limits of normal. Obviously in such instances the earlier figures do not represent basal conditions and are usually caused by failure of the patient to completely relax. It is most important in the performance of this test that the patients be under basal conditions, otherwise the results are of no value. The candidates for this test are often nervous and apprehensive and hence at times are with difficulty at the first test brought under basal conditions. Ziegler and Levine[6], Landis[7], and Henry[8] have demonstrated that mental unrest during the procedure can cause an elevated metabolism as high as +40. To overcome the various possibilities of error and to avoid accepting the results of a single test as confirmation of a diagnosis of thyroid toxicity, a safe rule to follow when the reported findings are elevated is to repeat the test until normal figures are obtained or until the rate reaches a stationary level. Figures reported up to +15 should be considered within the limits of normal especially in ambulatory patients[9].

The following case histories illustrate the manner in which repeated metabolic studies will finally clear up the diagnosis:

Case IV. A woman, aged 58, had suffered from pains in her legs for several months. A moderately severe grade of diabetes was present, requiring insulin. For some time there had been rather marked nervous and mental symptoms. Feeling that a thyroid adenoma might be the cause of her symptoms, a metabolic test was made. The rate was found to be +26, three days later +24, and on the next day +4. After eighteen months of insulin treatment the patient was found to be greatly improved in health, particularly in respect to the nervous and mental symptoms.

Case V. A woman, aged 30, had complained of nervousness and rapid heart action for several weeks. There was no loss of weight, sensitiveness to heat or thyroid enlargement. An internist, feeling that a diagnosis of hyperthyroidism should be considered, ordered a metabolic study. The rate on December 14 was found to be +27; and one week later, +27. She was then admitted to the hospital for further study. Here the metabolic rate was found to be, on January 9, +37; on January 10, +26; and on January 17, +13, which is within the limits of normal. She was discharged somewhat improved with a diagnosis of a neurosis. A rate taken 6 months later gave a figure of +3; all symptoms were relieved

and there had been a gain of 15 pounds in weight. It would have been very easy to have made the error of removing the thyroid here if repeated metabolic tests had not been made.

Case VI. A nurse, aged 21, with previously good health, after taking over the responsibilities of a ward supervisor, developed fatigue, nervousness, rapid heart action, anorexia and some loss of weight. The possibility of the presence of hyperthyroidism was considered. The metabolic rate on May 3 was found to be +26; on May 8, +34, and after a brief rest from her duties the rate was found to be +11. Because of the presence of some nervous and gastric symptoms her vacation was extended for two months, after which she was feeling in the best of health and her weight had increased by 20 pounds. At this time the metabolic rate was found to be +8. Two years have now elapsed with no return of symptoms. Here again repeated metabolism tests cleared up the diagnosis and saved the patient from unnecessary surgery.

The question often arises whether or not hyperthyroidism is ever present when the metabolic rate is normal. The majority opinion, especially of those having extensive experience with the test, is that this is uncommon—perhaps even less common than the presence of syphilis when the Wassermann test is negative. One must be cautious of accepting relief from surgery as proof of the presence of hyperthyroidism. The neuropathic patient is commonly relieved at least temporarily by any surgical procedure but time will eventually bring out the same or a new train of symptoms. At times the removal of an adenomatous or nodular goiter will give marked relief in the presence of a normal metabolism. In such cases the mechanical relief must not be lost sight of and the normal metabolism may be explained by the taking of iodine or the presence of a remission period so common in this type of struma. The socalled burned out toxic goiter usually has a normal or near normal rate, but shows extensive clinical evidence of damage due to long standing toxicity.

Hyperthyroidism for the most part is a well defined, yet often not easily diagnosed symptom complex. The most dependable symptoms are (1) definite loss of weight in the presence of a normal or increased food intake, (2) a persistent tachycardia of over 80, (3) a constant feeling of warmth or a sensitiveness to heat. On the other hand symptoms often associated with Graves' disease such as fatigue, sweating, tremor, transient rapid heart rate, choking sensations, loss of weight in the presence of anorexia and subnormal food intake, nervousness and emotionalism are more often caused by functional disorders of the nervous system than by true thyroid disease. It should always be kept in mind when a goiter is present that nervous symptoms may at times be present and be unrelated to it.

Care should be exercised in diagnosing mild hyperthyroidism with metabolic rates of from +15 to +35 especially if of short duration. These cases should be kept under observation for a time and repeated metabolic studies made. Ultimately normal figures will be obtained in the great majority of these border line cases as is well illustrated in cases V and VI.

The presence of mental symptoms, hysterical seizures, fits of depression, crying spells and marked emotionalism should be a warning that one may not be dealing with thyroid disease. This is well illustrated in case III. Like-

wise marked chronicity of nervous symptoms, especially where there is a marked nervous or mental family tendency, is a factor against the diagnosis of thyroidism.

Great caution should be exercised in making a diagnosis of hyperthyroidism in a patient under age 20. Because of the presence of a struma and certain nervous symptoms, the error is often made. The metabolism in this group is often found to be below normal pointing rathe rto a hypofunction of the thyroid. Surgery is rarely indicated and then only after most careful consideration of the diagnosis.

Summary

1. The term Graves' disease, because of its association with the thyroid gland, should be reserved for cases of hyperthyroidism.

2. The large neurogenetic group simulating hyperthyroidism and usually classified under the term Graves' diease should be designated by some other term to indicate its neuropathic origin and to avoid any association with the thyroid gland.

3. The basal metabolism test is the most important factor in the diagnosis of hyperthyroidism, when properly performed and interpreted.

4. The most dependable symptoms of hyperthyroidism are persistent tachycardia, loss of weight in the presence of a normal or increased food intake and a sensitiveness to heat.

5. Hyperthyroidism should be diagnosed with caution under the following conditions; in the presence of mental symptoms, when symptoms are mild and the metabolic rate is from +15 to +35, when there is marked chronicity of nervous symptoms and in cases under age 20.

REFERENCES

[1]Kessel, L.; Lieb, C. C., and Hyman, H. T.: A study of exophthalmic goiter and the involuntary nervous system, Jr. Am. Med. Assoc., 1922, lxxix, 1213-1216.

[2]Moschcowitz, Eli: The nature of Graves' disease, Arch. Int. Med., 1930, xlvi, 610-629.

[3]Hamilton, B. E., and Lahey, F. H.: Differentiation of hyperthyroidism and heart disease from neurasthenic states, Jr. Am. Med. Assoc., 1922, lxxviii, 1793-1796.

[4]Benedict, F. G.: The measurement and standards of basal metabolism, Jr. Am. Med. Assoc., 1921, lxxvii, 247-250.

[5]DuBois, E. F.: Basal metabolism in health and disease, 1924, Lea and Febiger, Philadelphia, p. 104.

[6]Ziegler, L. H., and Levine, B. S.: The influence of emotional reactions on basal metabolism, Am. Jr. Med. Sci., 1925, clxix, 68-76.

[7]Landis, C.: Studies of emotional reactions; metabolic rate, Am. Jr. Physiol., 1925, lxxiv, 188-203.

[8]Henry, G. W.: Basal metabolism and emotional states, Jr. Nerv. and Ment. Dis., 1929, lxx, 598-605.

[9]Barach, A. L., and Draper, G.: The significance of the basal metabolism determination, Jr. Am. Med. Assoc., 1925, lxxxiv, 740-745.

Hero Worship and the Propagation of Fallacies

(Lessons from the Lives of Jean Astruc and John Hunter)*†

By C. S. Butler, M.D., F.A.C.P., Captain, (M.C.), U.S. Navy,
U.S. Naval Hospital, Brooklyn, N.Y.

GABRIEL LeClerc in the year 1692 wrote a compend entitled "La Chirurgie Complete" which, according to Garrison went through eighteen editions. The fourth edition was translated into English in 1707, and we will quote part of the title-page of this because of its pleasing quaintness of diction: "The Compleat Surgeon: or the Whole Art of Surgery explain'd in a most familiar method. Containing an exact account of its principles and several parts, viz. Of the bones, muscles, tumours, ulcers, and wounds, simple and complicated, or those by gun-shot; As also of Venereal *Diseases*, the Scurvy, Fractures, Luxations, and all sorts of Chirurgical Operations. To which is added a Chirurgical Dispensatory; shewing the manner how to prepare all such Medicines as are most necessary for a Surgeon, and particularly the Mercurial Panacaea." This must have been a popular compendium of surgery for the students of those days both French and English. The delightful little book gives an excellent discussion of the venereal diseases, correct as to types with the omission of chancroid which was not defined until 1858. Gonorrhea or the "Chaudepisse" and its complications and syphilis, or the pox, were handled much as they are today. Indeed the treatment of these two diseases is about as effective as it was at the beginning of the twentieth century.

Jean Astruc (1684-1766), celebrated physician of Louis XV was so scholarly at the height of his career that few contemporaries dared oppose him. In addition to writing a critique upon certain parts of the Bible, he gained for himself a well-earned reputation as a literary physician at a time when medicine was emerging from Medievalism. Of his medical works, perhaps the most important was entitled "A Treatise of *the* Veneral Disease in Six Books". The Latin text of 1736 was translated into English the following year. It has never been understood even by his own countrymen how such a scholar as Astruc could lend himself to the propagation of so much that was fallacious in a single volume. As is well known, he contended that there was only *one* venereal disease and that this had been transferred from America to

*Read at the Annual Meeting of the American Society of Tropical Medicine, New Orleans, Louisiana, November 20th, 1931.

†Received for publication, November 2, 1931.

Europe by Columbus' ships. The whole force of his classical education was exerted to prove these two fallacies forty-four years after LeClerc's first edition, and though his writings were cried down by other scholars of the day, yet it is a matter of history that his work on "the venereal disease" has influenced medical thought and literature down to the present time. So much of an impression did he make upon the medical men of his time and succeeding generations that it requires only a perusal of the volume of John Hunter entitled "A Treatise on *the* Venereal Disease", published in 1786, to see that Hunter not only embraced Astruc's views, but actually the title for his own work. He accepted Astruc's interpretation of the venereal diseases in toto. He puts aside, however, the American origin of syphilis with a sentence, in which it appears that he does not care to consider this feature, though agreeing with Astruc as to the appearance of syphilis in Europe at a recent date.

As to the question of the viruses of gonorrhea and syphilis being the same, Hunter was so positive of the truth of this that in 1767, when he was 39 years of age, he inoculated himself with material from what was probably a urethral chancre though he thought it gonorrhoeal pus. Born in 1728, he died in 1793, and it was evident from his description of his own symptoms and from the write-up of Sir Everard Home on the course of the disease from which he died, that this inoculation influenced his entire future career. Hunter's first attack of angina pectoris occurred in 1773, just about six years after his unfortunate inoculation. By 1785 he had progressed so far that generalized arteriosclerosis had extended to the point of producing cerebral symptoms, due to arteriolar spasm simulating the Adams-Stokes syndrome. Following a period of unconsciousness, he had twitchings of the muscles of the nose, face, lower jaw, and left arm. This was a fairly typical example of the syphilitic cerebral syndrome. By the year 1789 a type of oculomotor palsy had developed which was most likely syphilitic. Few histories are more pathetic than that of the devotion of this great man to a search for the facts about syphilis. The influence of Astruc, however, is so evident in his case that it needs only a perusal of the two volumes to see that Astruc's views held him captive. Hunter's works are among the great contributions to English medical literature. In turn his force and personality and practical knowledge won many generations of physicians who in turn accepted his word as law.

In a delightful study of the history of certain medical instruments Professor Logan Clendening[1] describing the revolution which Schönlein and his students effected in the teaching of clinical medicine uses the following phrase: "Happy is that medical school which has on its faculty three or more intensely vivid clinical teachers who hate one another and despise their rival's methods and views. Stimulating is the mental atmosphere of such an establishment." Schönlein, Frerichs, Traube, and Wunderlich in a very brief period revolutionized clinical teaching and gave to medicine a proper estimate of thermometric de-

terminations in disease and of the importance of temperature curves in various infections. The brief history of these two groups shows the relative value of hero worship and of intellectual competition in the search for truth.

In 1816 another great French physician was at the height of his professional career, Doctor A. J. L. Jourdan. In that year he published in the "Journal Universel des Sciences Médicales" five articles entitled "Considerations Historiques et Critiques sur la Syphilis". Jourdan, like Astruc, was also a medical *literateur*, but Astruc had some seventy-five years advantage in point of time. In these articles Jourdan completely disproves Astruc's ideas in regard to the origin of syphilis, and backs it up with incontrovertible evidence. This gem of medical literature was translated into English by Doctor R. LaRoche in 1823 and published in Philadelphia in that year. It has remained buried there for the past century. In 1826 Jourdan wrote his exhaustive treatise entitled "Traité Complet des Maladies Veneriennes", 2 volumes, Paris, in which he gives all the material concerning the subject under consideration which had come out in the first named publication. Opinions and facts we think quite modern were shown by this French scholar to extend well into the Dark Ages and in some cases into ancient times. While his views upon constitutional syphilis were incorrect, most of his work was sound. In spite of these and other more recent treatises showing the fallacies in Astruc's work, one of these fallacies goes merrily on and at the present time is held by the majority of physicians of Europe and America. We refer to the American origin of syphilis which Astruc appropriated from Oviedo's book of 1525.

Coming back to Hunter, whose opinions were now considered as infallible, we find that he ventured to express positive views upon matters about which he knew little and to indulge in speculation in a way not calculated to advance a knowledge of medicine. We are speaking now of Hunter, the syphilographer. As a urologist, Hunter was years in advance of his time.

On pages 14 and 15 of his treatise, he attempts to show how "the venereal disease" was introduced into the South Seas. Calling upon the voyages of Captain Cook, who, as is well known, visited most of the island groups of the South Pacific between the years 1768 and 1779, he expressed the view that it was the French under Bougainville, 1766 to 1769, who first introduced the venereal disease into Otaheite (Tahiti) for although Cook visited Tahiti sometime in advance of Bougainville, Hunter convinces himself that it was Bougainville who introduced the venereal diseases into the Society group. As a matter of fact both the English and French fleets were important factors in spreading venereal diseases through the islands of the South Pacific. In connection with Hunter's statements in this regard, we will quote the views expressed by one or two modern writers upon tropical medicine. Manson-Bahr[2] expresses himself upon the origin of yaws in the following terms: "The home of yaws at the present day is within the true tropics, between

Capricorn and Cancer; its chief ravages are mainly confined to the old world; its distribution in the new (West Indies, Venezuela, Guianas, and Brazil) being directly due to the infamous influence of the slave trade. Thus has Fate decreed that the gift of syphilis from the new world to the old, consequent upon the Spanish Conquest, should be repaid in kind some hundred years later by the exportation of yaws from Africa by negro slaves." In "Tropical Diseases" by the same author[3], occurs this sentence in connection with immunity in yaws: "Apparently saturation of a community with yaws virus produces a relative immunity to syphilis. On these grounds may be explained the apparently well authenticated fact that syphilis is *absent* amongst the Polynesians of Fiji, Tonga, and Samoa, in whom yaws is especially prevalent." Now it is axiomatic in the epidemiology of syphilis that there is no place on earth the white man has visited from which syphilis is *absent,* so that Manson-Bahr is but expressing the views of Astruc and Hunter, views of the middle of the eighteenth century, when making these statements in 1928 and 1929. Hunter's remarks upon the origin of "the venereal disease" in the South Sea Islands is but another example of the unscientific, not to say ungenerous, effort to "wish" syphilis off on the people of other races, efforts so common in the fifteenth and sixteenth centuries.

On pages 382 and 383 of the 1786 edition, Hunter speaks with assurance upon yaws. It appears that he saw only one case of this condition in the course of his career and upon this he makes his positive statements. In this he reminds us very forcibly of certain "library authorities" on yaws at the present time. At this place in his treatise we find such assertations as the following: "Yaws have a regular progress after going through which they leave the constitution in a healthy state at least free from that disease. It being sufficient for the cure that the patient be in a state favorable to general health." This resembles very much some of the 1930 assertions emanating from Manila, that yaws is one of the easiest of all constitutional diseases to cure, assertions made in complacent disregard of the controverting experience of scores of tropical practitioners in other parts of the world. However, I think that Berkeley Hill's sentence about Hunter's *one* case of yaws proves the soundness of someone's belief in the emphasis of understatement. Here is Hill's sentence (Syphilis and Local Contagious Disorders, page 15): "John Hunter writing 'on diseases resembling the lues venerea, which have been mistaken for it', describes a case of yaws that was clearly syphilis, for the very reasons he advances to prove it could not have been that disease." Castellani and Chalmers[4] writing upon yaws make the statement that the endemic home of yaws is in America and that Sydenham's statement that it is in Africa is not compatible with the facts as now known. "It must be remembered", they remark, "that the discovery of America led to a great many voyages in all directions, and we need not be surprised to find that Bontius in 1718 found the disease to be endemic in Java, Sumatra, and other

Dutch Colonies in the East Indies.... In 1832 Bennet stated that it was endemic in the Tonga, Society, and Navigator Islands, while Koeniger believes that its introduction into the Samoan group was comparatively recent. It is therefore possible that it may have spread throughout the tropics from America, both in an easterly and westerly direction." Was it the white crews making the voyages "in all directions" that spread yaws to the Orient? Yaws is *not* a white man's disease. The date, 1832, is over 50 years after Bougainville and Cook. Thus do the authorities on tropical medicine differ very markedly on the geography of yaws and here they are accepting the views of the older writers who knew much less about the facts than we of the present.

There is no way of reconciling the diverse statements and opinions expressed by John Hunter and the others quoted with the facts as known about yaws. Such bizarre, fanciful statements are made by the "yaws experts" in all parts of their description of it from definition to treatment! These fanciful statements make of this condition what we have elsewhere spoken of as a medical monstrosity. The fallacies of these statements have been considered by many writers for all of the divisions of the description of this *impossible* entity. We should like to notice only one more of these inconsistencies having to do this time with the pathological side of the description of yaws. In an article entitled "Yaws, As Observed in Haiti", Doctor Howard Fox[5] quotes from the laboratory report the following pathological differentiation: "Microscopic examination of early and late yaws showed an edematous verrucous plasmoma in which the infiltration was horizontally delimited below and was not intimately related to inflamed blood vessels, as in syphilis. Plasma cells were more succulent and larger than in syphilis, giant cells were rare and there was no productive inflammation of the blood vessels." Most of these statements have been disproven or properly evaluated by Choisser and others, but it seems that the pathologist here depends upon the relative "succulence" of the plasmoma cells to enable him to differentiate the histopathological picture of yaws from that of syphilis. One wonders if a "standard of succulence" might not be established in order to aid in such differentiation. The description of the histology of the yaws granuloma harks back to the erroneous conclusions of MacLeod[6], who compared the framboesioma, *not* with its luetic reflected image, the condyloma and the circinate syphilid, but with the histological picture of syphilides in general. Moreover, MacLeod seems to have labored under the impression that framboesia did not affect the viscera. This is quite erroneous. At the present time there is a considerable amount of autopsy material from cases of Haitian treponematosis being studied in the Department of Pathology of the University of Michigan and it is hoped that this investigation will give us the facts about the histopathology of yaws.

In conclusion, we may say that the effect of the work of Astruc and Hunter upon our knowledge of the venereal diseases has been to confuse

rather than to clarify, to perpetuate fallacy rather than to uncover truth. A comparison of their careers with those of Schönlein, Frerichs, Traube, and Wunderlich leads to the conclusion that in advancing a knowledge of medicine it is better to have open intellectual combat than to have too much scholarship and too much hero worship. It has fallen to the speaker's lot to be unable to accept some of the current views regarding yaws and syphilis. It is not a pleasant task to be an "image breaker" in this field. Carlyle did not treat of the "Hero as Physician." Had he done so, he might have found several candidates for Hero Worship in tropical medicine. It is our firm belief that when the blight of hero worship is eradicated from tropical medicine, the views of Hutchinson and of Jourdan will prevail.

REFERENCES

[1]Clendening, L.: History of certain instruments, Ann. Int. Med., 1930, iv, 176-189.

[2]Manson-Bahr, P. H.: Yaws, Brit. Jr. Ven. Dis., 1928, iv, 44-54.

[3]Manson-Bahr: Tropical diseases, 1929, William Wood and Company, New York, p. 500.

[4]Castellani, A., and Chalmers, A. J.: Frambesia tropica, (Yaws), The practice of medicine in the tropics, Byam and Archibald, 1922, ii, Henry Frowde and Hodder and Stoughton, London, p. 1309.

[5]Fox, H.: Yaws (frambesia tropica) as observed in Haiti, Arch. Dermat. and Syph., 1929, xx, 820-834.

[6]MacLeod, J. M. H.: Contribution to the histo-pathology of yaws, Brit. Med. Jr., 1901, 797-803.

Editorial

THE JOHN PHILLIPS MEMORIAL PRIZE

It is with very sincere pleasure, indeed, that announcement is made elsewhere in this issue, in the form of an excerpt from the Minutes of the December meeting of the Regents of the American College of Physicians, of the award of The John Phillips Memorial Prize to Doctor Oswald T. Avery. This award is in recognition of the work of Dr. Avery on the capsular polysaccharides of the pneumococci, leading to the production of an enzyme having a specific and selective action upon the Type III polysaccharide, both in the chemically pure state and as it exists in the capsules of the living pneumococci. Further, Dr. Avery has shown that mice, injected with an active preparation of the enzyme together with multiple lethal doses of a virulent culture of Type III pneumococcus, survive infection. The specific enzyme, by decomposing the capsular polysaccharide, apparently renders the infecting organism vulnerable to the defensive mechanisms of the animal body. Thus a new mode of attack upon the causal agencies in the infectious diseases is opened for experimental investigation.

THE LYMPHOID TISSUE CHANGES IN THE PRODROMAL STAGE OF MEASLES

In a paper which was not published until after his death, Warthin[1] described the occurrence of large multinucleate giant cells in the tonsils of four patients who subsequently developed measles. In each instance, as afterwards appeared, tonsillectomy had been performed from twenty-four to ninety-six hours before the appearance of the exanthem. So characteristic were the histopathological findings that they were used successfully in the later cases of the series to predict the onset of measles before clinical evidence was available. One child was removed from an open ward and placed under contagious disease precautions on the strength of the pathological diagnosis alone, and the subsequent appearance of the eruption justified the confidence which the clinician had placed in the diagnosis made by the pathologist. The essential pathological lesion in the tonsils and pharyngeal mucosa in the prodromal stage of measles was found to be a subepithelial infiltration of multinucleate giant cells, lymphocytes and monocytes, with wandering of the giant cells into the mucosa and on to its surface. The germinal centers of the lymphoid

[1] WARTHIN, ALDRED SCOTT: Occurrence of numerous large giant cells in the tonsils and pharyngeal mucosa in the prodromal stage of measles, Arch. of Path., 1931, xi, 864-874.

follicles were found to show lymphoid exhaustion and here, also, numerous multinucleate giant cells were present. A cervical lymph node from one of these patients, examined after recovery from measles, showed no similar changes. That these changes are not confined to the tonsils and pharyngeal mucosa is evident from a recently reported observation of Herzberg.[2] On the fourth day after an appendectomy a child developed a rash on the face, body, and extremities, with Koplik spots, conjunctivitis and cough. A definite diagnosis of measles was made. In several of the lymph follicles of the appendix, but apparently in a sharply localized area only, there were found multinucleate giant cells. From the description and illustrations of these giant cells and the associated hyperplasia and lymphoid exhaustion of the germinal centers, it seems certain that Herzberg has found in the appendix during the prodromata of measles the same lesion which Warthin found to be pathognostic of the onset of this disease when seen in the pharyngeal mucosa and tonsils. This provides evidence of a more general invasion of the lymphadenoid tissues during the onset of measles than had hitherto been demonstrated. It is to be hoped that others will search for these apparently highly significant histological changes when opportunity is presented.

[2]Herzberg, Mortimer: Giant cells in the lymphoid tissue of the appendix in the prodromal stage of measles: report of an isolated case, Jr. Am. Med. Assoc., 1932, xcviii, 139-140.

PROGRAM OF THE SAN FRANCISCO MEETING

In the College News Notes section of this issue will be found the final program of both the General and the Clinical Meetings of the Sixteenth Annual Clinical Sessions of the American College of Physicians. Fellows and Guests expecting to attend will find it highly advantageous to observe the directions which are included having to do with transportation, reservations, and registration for Clinics and Demonstrations. In richness of material, breadth of interest and skill in arrangement, these programs give evidence, on the one hand, of the unselfish labors of those whose task it was to assemble them, and, on the other, of the great wealth of material for didactic and clinical medicine which San Francisco and its Pacific Coast neighbors possess. An unusually profitable meeting is in prospect for all who can attend.

Abstracts

The Detection and Estimation of Radium in Living Persons. III. The Normal Elimination of Radium. By HERMAN SCHLUNDT and G. FAILLA. (Am. Jr. Roentgenol. and Rad. Therap., 1931, xxvi, 265-271.)

Using as subjects two girls who had contracted radium poisoning during employment as dial painters during the period 1917 to 1919, the authors determined the rate of elimination of radium by determining the total quantity of radium present in each subject, as well as the quantity eliminated each day. The first of these values was arrived at by making gamma-ray measurements on the subjects by means of a standardized electrometer and adding to the value thus obtained the quantity of radium responsible for the radium emanation (radon) found present in the air expired. Combining all the experimental results, it appeared that the radium content of Subject A, twelve years after exposure, was 24 μg; and of Subject B, 14 μg. The quantity of radium removed day by day by excretion was determined by estimations, about 90 per cent of the amount eliminated being found in the feces. The total radium eliminated daily by Subject A amounted to 1161 $\mu\mu$g, corresponding to a coefficient of daily elimination of 0.005 per cent. The total daily elimination of Subject B was 363 $\mu\mu$g., giving a coefficient of daily elimination of 0.0026 per cent. Possible reasons for the difference in the coefficients for the two subjects are discussed. It is of interest that reduction by the process of natural decay of the element need not be considered practically in the case of radium stored in human body, for its half period is about 1600 years. The experiments reported show, therefore, that radium continues to be eliminated even twelve years after ingestion, although the rate of elimination is very slow. They also show that most of the radium eliminated is found in the feces. The results throw considerable doubt upon the validity of the assumption that the stored radium is distributed uniformly throughout the skeleton.

Involvement of the Central Nervous System in a Case of Glandular Fever. By SAMUEL H. EPSTEIN, M.D., and WILLIAM DAMESHEK, M.D. (New England Jr. Med., 1931, ccv, 1238-1241.)

Severe headache, blurring of vision, photophobia, and general malaise had marked the onset of disease in a young man, 19 years old, who was admitted to the hospital in a stuporous condition. The spleen could be felt and there was a generalized enlargement of the lymph nodes. The total white blood cell count was 14,900 per cubic millimeter, with 63 per cent lymphocytes and 8 per cent monocytes. In all respects the morphology of the blood was typical of that occurring in infectious mononucleosis. Lumbar puncture yielded a clear, colorless fluid under normal pressure. On admission there were 34 white cells per cubic millimeter, chiefly lymphocytes. This number rose to 44 eight days later, but after that time gradually decreased, falling about proportionately with the decrease in white cells in the blood. Culture of the spinal fluid showed no growth and the Wassermann reaction was negative. The subsequent clinical course, resulting in complete recovery, served to rule out certain possibilities in the differential diagnosis, particularly acute lymphatic leukemia and tuberculous meningitis. It appears obvious that in this case the acute neurological condition and the general lymphoid hyperplasia with mononucleosis must be closely related. The nature of this relationship cannot be answered completely at present. The close parallelism between the cellular changes in the cerebrospinal fluid and the changes in the leukocyte count is significant. The simultaneous clinical recovery and the return to normal of the white blood cells indicate at least a temporal relationship so that it is not

unreasonable to suppose that the same infectious agent which caused the lymphoid hyperplasia also produced the central nervous system condition. Johansen has recently reported a similar case emphasizing the relationship between serous meningitis and infectious mononucleosis.

Renal Lesions in the Toxemias of Pregnancy. By E. T. Bell, M.D. (The Am. Jr. Path., 1932, viii, 1-41; 3 plates.)

Twenty cases of toxemia of pregnancy were divided into five groups: (1) typical eclampsia with convulsions; (2) eclampsia without convulsions; (3) pre-eclampsia; (4) hyperemesis gravidarum, and (5) pregnancy in association with pre-existing renal disease. The pathological changes found in these patients at autopsy are detailed with special attention to the kidneys. In fatal cases of eclampsia and pre-eclampsia a characteristic glomerular lesion was found. The glomeruli were found to be slightly enlarged and the lumina of their capillaries narrowed. The decrease in size of the capillary lumina is caused chiefly by a marked thickening of the capillary basement membrane, but sometimes by an increase of endothelial cells. In one patient, who had had an attack of eclampsia seven years before, focal hyaline areas were found in the glomeruli with partial or complete glomerular obliteration and varying degrees of tubular atrophy. In one case of hyperemesis gravidarum, glomerular lesions were found like those of typical eclampsia. In three other cases the glomeruli were normal. A fatty liver without necroses is characteristic of this form of toxemia. When a woman with chronic renal disease becomes pregnant, there is usually an aggravation of all the nephritic symptoms. Chronic nephritics show no special tendency to develop gestation eclampsia.

The Influence of Solar Rays on Metabolism, With Special Reference to Sulphur and to Pellagra in Southern United States. By James H. Smith, M.D. (Arch. Int. Med., 1931, xlviii, 907-1063.)

Sulphur in the form of cystine appears to exert a protective action against exposure to solar radiation in low forms of life. Its high concentration in the epidermal tissues of higher animals suggests a possible protective action here also. Thus it appears that an adequate supply and a normal metabolism of sulphur exert a preventive influence against the pathologic effects of solar irradiation. Conversely, it is suggested that an inadequate supply of sulphur as cystine is an important cause of pellagra. Further, the distribution of pellagra and the variations in its prevalence and incidence suggest that solar irradiation, under certain abnormal conditions of nutrition, is an important factor in the etiology of pellagra, and that the reaction to solar rays not only is conditioned by the nutritive state, but depends on a state of the tissues determined by contrasts in degree and intensity of exposure during the annual cycle.

A Clinical Study of Myxedema in Michigan. By H. H. Riecker, M.D. (Jr. Mich. State Med. Soc., 1931, xxx, 831-835.)

From a study of the geographical distribution of 64 cases of myxedema in the series studied, and in comparison with the usual clinical experience in other regions, it is concluded that this disease is more common in the Great Lakes goiter district than in nongoitrous regions. In the series studied, 75 per cent of the patients were female; and 34 of the 64 were in the age-group 41-60. In 40 per cent the basal metabolic rate was between 15 per cent and 25 per cent below normal. The most common presenting symptom of the patients in this group was weakness. One organ or system was frequently noted to present outstanding signs or symptoms. Anemia, skin lesions, mental and cardiac complaints, and digestive disturbances illustrated this point. Obesity emphasizes rather than submerges the characteristics of the disease.

Über vorübergehende Hemiplegien durch Nicotin [*Transitory Hemiplegia Due to Nicotine*]. By F. Külbs. (Klin. Wochenschr., 1931, x, 2159-2161.)

In four cases, three men whose ages were between 21 and 38 years and a woman 40 years old, transitory paralytic phenomena appeared. In the three men these consisted of hemiplegia and speech disturbances. In the woman there was a motor aphasia. These

disturbances had been preceded by evidences of lessened functional capacity as shown by ease of fatigue, or by headache, paresthesia and dizziness. In all cases there had been gross overuse of tobacco, evidence of which was found in lymphocytosis, elevated basal metabolic rates and marked nervous and vasomotor irritability. Since there was a complete restoration to normal in all cases within a short time of the interdiction of the use of tobacco, it is believed that the hemiplegic phenomena were due to a vasoconstrictor effect of tobacco. Acute fatigue, hereditary factors and syphilis could all be excluded with certainty as etiological agents. This observation may be a significant contribution in respect to that group of cases which shows hemiplegia clinically, without anatomical changes adequate to explain its occurrence.

Reviews

Recent Advances in Allergy (Asthma, Hay-Fever, Eczema, Migraine, etc.) By GEORGE W. BRAY, M.B., C. M. (Sydney) Asthma Research Scholar, The Hospital for Sick Children, Great Ormond Street, London. With foreword by ARTHUR F. HURST, M.A., M.D. (Oxon), F.R.C.P., Senior Physician, Guy's Hospital; Chairman Medical Advisory Committee, Asthma Research Council of Great Britain. 432 pages; 98 illustrations, including 4 colored plates. P. Blakiston's Son & Co., Inc., 1012 Walnut St., Philadelphia, 1931. Price, $3.50.

The author's two years of research in asthma at the Hospital for Sick Children have evidently given him a wide experience with allergic disease. He reviews the important theories and surveys the large field of experimental work. Brief historical sketches are also interspersed. He discusses heredity and the endocrines as factors in the causation of allergy. The more usual manifestations of allergy, as asthma, hay-fever, eczema, urticaria and angioneurotic edema are taken up in some detail. Numerous other conditions, such as dermatitis venenata, migraine, epilepsy, muco-membranous colic, vaccine therapy, drugs, serum reactions, hypersensitiveness to insects, the effects of molds and fungi, are discussed as to their known and possible relation to allergy. Addition and elimination diets are given. This book gives a fairly complete treatment of the subject, written in a compact form, and in a clear, easily read style. Unfortunately, all pollination data are for the British Isles. So significant is geographical distribution and pollination chronology in the botanical aspect of allergy, that it would be well to have an American edition when reprinting is undertaken.

The Human Voice. By LEON FELDERMAN, M.D., x + 301 pages, 22 figures. Henry Holt and Company, New York City, 1931. Price, $2.50.

There is probably no medical book more difficult to write than that which endeavors to expound medical matters to lay readers. And there is no book which requires any greater degree of precision and accuracy in statement, for it must be assumed that the group for whom it is intended will be altogether unable critically to evaluate it. It is stated in the book under review that it is intended to bridge the difficulties between the vocal teacher and his pupil and that "a sincere effort has been made to strip discussions of technical terms and to write in a language familiar to all." In regard to the sincerity of the author and the innocence of the publishers there can be no question. The introduction of such terms as [in the actual spellings used] archi pallium, kinaesthetic, laryngo-periskop, pharmacotoxic, lancelating, streptococcic hemolyticus, and non-memolytic, leaves some doubt as to whether the language is understandable by all. More serious are actual errors in fact, stated or implied. Some of these are of an extraordinary nature. For instance, in regard to the possible spread of syphilis by unsanitary lunch rooms and soda fountains, it is stated that: "Hordes of flies gather, ready to carry the *larvae* from one place to another, and the vicious circle of

contagion is maintained." The combination of syphilis as a fly-borne disease and of the Treponema as a larva in the same sentence compels a pause even from one who is accustomed to grading examination papers. The author is evidently a believer in the efficacy of maternal impressions in the etiology of disease, for he lists among the causes of stammering, a fright or a profound disappointment experienced by the mother during the prenatal period. Scientific medicine has been striving for several decades to release the human mind from the bondage of such erroneous ideas. Thus the reviewer is forced to the conclusion that to be both useful and safe this book requires extensive revision.

Confessio Medici. By STEPHEN PAGET, F.R.C.S., Late Vice Chairman Research Defense Society. xi + 158 pages. The Macmillan Company, New York City, 1931. Price, $2.00.

Again these charming essays are made available for the general medical reader. Published in 1908, this reissue brings them to a new generation of physicians. They can never grow old for they are of the medical life itself. Vocation, Hospital Life, An Essay for Students, A Good Example, Practice, The Discipline of Practice, The Spirit of Practice, Wreaths and Crosses of Practice, Retirement, and The Very End are the titles of the series, with a Preface and an Epilogue. Since each essay is complete in itself this is a book to be picked up in the moment of leisure when the available time is short or to be taken along as a companion on a journey. The publishers are to be commended for bringing out this new issue.

Guide to Radiologic Diagnosis in Heart Disease. Prepared with the Aid of the Committee on Research of the Heart Committee. By GEZA NEMET, M.D. 33 pages, 31 figures. New York Tuberculosis and Health Association, Inc., 386 Fourth Avenue, New York City, 1931. Price, 35 cents.

A concise system for the interpretation of roentgenograms of the heart is set forth in the interest of securing suitable technical procedures and a more uniform nomenclature.

Criteria for the Interpretation of Electrocardiograms. Prepared with the Aid of the Committee on Research of the Heart Committee. By ARTHUR C. DEGRAFF, M.D., 19 pages, 42 figures. New York Tuberculosis and Health Association, Inc., 386 Fourth Avenue, New York City, 1931. Price, 35 cents.

This is a concise statement of criteria in electrocardiography arranged by diagnoses and by terminology.

College News Notes

THE PHILLIPS MEMORIAL PRIZE

The following announcement is an excerpt from the minutes of the meeting of the Board of Regents held December 20, 1931:

"Dr. Means, Chairman of the Phillips Memorial Prize Committee, reported that his Committee had proceeded according to the revised regulations adopted by the Board of Regents, which were, briefly, that the Committee seek a candidate for the prize rather than invite submission of manuscripts as had been done the year before. After discussing the procedure of his Committee, he presented the name of Dr. O. T. Avery, Hospital of the Rockefeller Institute for Medical Research, New York City, with the recommendation to the President that the prize be awarded to Dr. Avery 'for the series of studies upon the pneumococcus in which he has played a leading rôle, beginning with the discovery of the type specific soluble capsular polysaccharides and culminating in the discovery of a bacterium which produces an enzyme which splits the polysaccharides of Type III pneumococcus in vitro, thus rendering it susceptible to phagocytosis and thereby protecting the animals which are infected with it.'

"This recommendation was adopted unanimously.

"President White stated that if it could be arranged, Dr. Avery's presentation should be made at the time of the Convocation, and preceding the Presidential Address, to which the Board agreed."

Acknowledgement is made of the receipt of gifts to the College Library of publications by members, as follows:

Dr. Henry Daspit (Fellow), New Orleans, La., 1 autographed copy, No. 54, "Matas Birthday Volume";

Dr. Hyman I. Goldstein (Associate), Camden, N. J., 1 reprint;

Dr. H. Beckett Lang (Fellow), Marcy, N. Y., 5 reprints;

Dr. William D. Reid (Fellow), Boston, Mass., 3 reprints;

Dr. Walter M. Simpson (Fellow), Dayton, Ohio, 1 reprint, "Aldred Scott Warthin";

Dr. Charles T. Way (Fellow), Cleveland, Ohio, 1 reprint;

American College of Surgeons, 1 copy of 1932 Directory.

Dr. Frank Smithies (Master), Chicago, Ill., presented a medical clinic before the Mahoning County Medical Society at the Youngstown, Ohio, Hospital, October 20, and on the evening of the same date, addressed the Mahoning County Medical Society on "Gastric Hemorrhage."

Dr. Louis Faugeres Bishop (Fellow), New York City, as one of the Trustees of Rutgers University, has been assigned to the Governing Committee of the New Jersey College of Pharmacy, Newark, N. J. The College will give a four year course leading to the degree of B.S. in Pharmacy. The course will cover the requirements of the American Medical Association of a premedical course for those students who may wish later to study medicine.

Dr. D. N. Kremer (Fellow), Philadelphia, Pa., addressed the Bay Ridge Medical Society, Brooklyn, N. Y., December 8, on "Obesity and Its Management."

Dr. I. S. Kahn (Fellow), San Antonio, Texas, in collaboration with Dr. B. F. Stout of San Antonio, read a paper entitled, "The Practical Value of the Cytological Examination of the Nasal Smear in the Differential Diagnosis Between Allergy and Infection (Preliminary Report)," at the recent meeting of the Southern Medical Association.

Dr. W. J. Stapleton, Jr. (Fellow), Detroit, Mich., gave a lecture on "Medicine in Art" before the Detroit Medical Club on December 17, 1931.

Dr. Stapleton also gave a radio talk on the subject, "Periodic Health Examination" over Station WWJ, January 5, 1932.

Dr. Ralph Pemberton (Fellow), Philadelphia, Pa., recently gave a paper on "Arthritis" at the Annual Clinic of the Sectional Meeting of the Michigan State Medical Society at Jackson, Mich.

Dr. Horace W. Soper (Fellow), St. Louis, Mo., read a paper on "Roentgen Diagnosis of Lesions in the Small Intestine," with x-ray illustrations, before the Radiological Society of North America on December 1, 1931.

Medical Clinics by the following Fellows of the College were published in the December Number of the Hahnemannian Monthly:

Dr. G. Harlan Wells, Philadelphia, "Trichiniasis";

Dr. Carl V. Vischer, Philadelphia, "Bronchial Asthma";

Dr. Donald R. Ferguson, Philadelphia, "Chronic Valvular Heart Disease of Rheumatic Origin with Passive Congestion."

Dr. John R. Vonachen (Fellow), Peoria, Ill., was recently elected President of the Peoria County Medical Society.

Dr. Ada E. Schweitzer (Fellow), Indianapolis, Ind., Child Hygiene Director of the Indiana State Board of Health, has recently issued several interesting and helpful bulletins, promoting infant and child hygiene and health in that state.

Dr. Clyde L. Cummer (Fellow), Cleveland, Ohio, has recently published the third edition of "Manual of Clinical and Laboratory Methods," through Lea & Febiger, Philadelphia.

Dr. Cummer is the author of an article entitled, "Lupus Erythematosus in Infancy and Childhood," Archives of Dermatology and Syphilology, 1931, xxiv, 999-1032. This article was originally read at the meeting of the American Dermatological Association at Toronto during 1931.

Dr. Robert E. Ramsay (Fellow), Pasadena, Calif., has recently been elected President of the Pasadena Branch of the Los Angeles Medical Association.

Dr. Joseph H. Bryan (Fellow), Asbury Park, N. J., has been elected President of the Alumni Association of the New York Homeopathic Medical College and Flower Hospital. Dr. Bryan was a member of the graduating class of 1890 of that institution.

Dr. W. G. Herrman (Fellow), Asbury Park, N. J., read a paper entitled "Varieties of Pulmonary Spirochetosis" before the American Roentgen Ray Society, at their last meeting in Atlantic City. Dr. Herrman also published an article entitled, "Uterine Hemorrhage Radiologically Considered," in a recent issue of the Journal of the New Jersey State Medical Society.

Dr. Harold S. Davidson (Fellow), Atlantic City, N. J., has been elected President of the Atlantic County Medical Society for the year 1932.

Dr. W. H. Fairbanks (Fellow), Freehold, N. J., has been appointed a member of the Executive Committee of the Monmouth County Medical Society. He has been a member of the Board of Health of Freehold for five years.

Dr. W. G. Richards (Fellow), Billings, Mont., recently conducted for the Indian Department a tuberculosis survey of the Crow and Cheyenne Indians.

At the national meeting of the Milwaukee Academy of Medicine on January 12, the following Fellows of the College were elected:

Dr. R. W. Blumenthal, President;

Dr. C. H. Stoddard, Vice President;

Dr. Francis D. Murphy, Membership Committeeman;

Dr. J. Gurney Taylor, Chairman, Milk Committee.

Dr. Samuel A. Levine (Fellow), Boston, Mass., will be the Guest Speaker on a Symposium on Coronary Disease, in connection with the General Medicine Section of the California State Medical Association meeting at Pasadena during the first week of May.

Dr. R. Manning Clarke (Fellow), Los Angeles, Calif., is Secretary of this Section.

Dr. Hay M. Balyeat (Fellow), Oklahoma City, Lecturer on Diseases Due to Allergy, University of Oklahoma Medical School, addressed the Hot Springs Academy of Science at Hot Springs National Park, February 2, on the subject "Recent Advances in Allergy."

Dr. Wardner D. Ayer (Fellow), Syracuse, New York, was recently elected President of the Syracuse Academy of Medicine.

Dr. Earle E. Mack (Associate), Syracuse, New York, was recently elected Secretary of the Syracuse Academy of Medicine, and was also re-elected to the Secretaryship of the Onondaga (County) Medical Society.

Dr. Joseph H. Barach (Fellow) addressed the Valley Medical Society, Glassport, Pa., January 21, 1932. Dr. Barach's subject was "Clinical Interpretation of High Arterial Pressure."

OBITUARIES

DR. RALEIGH PETER HALE

Dr. Raleigh Peter Hale (Fellow), East Chicago, Indiana, died December 1, 1931, of heart disease; aged 48 years.

Dr. Hale was born at Columbia, Mo., attended Northwestern University, from which he received his medical degree in 1908. He was a member of the Indiana State Medical Association, and a past President of the Lake County Medical Society. He was elected a Fellow of the American College of Physicians on February 24, 1926.

DR. ADAM CLARKE DAVIS

Dr. Adam Clarke Davis (Associate), Creighton, Pa., died December 13, 1931; age, 62 years. Dr. Davis graduated from the University of Pittsburgh School of Medicine in 1894. He was a member of the Allegheny County Medical Society, the Medical Society of Pennsylvania and the American Medical Association. He became a member of the American Congress on Internal Medicine on March 13, 1925, by virtue of which membership he was transferred to Associateship in the College at the time of the merger of the two organizations during 1926.

PROGRAM SIXTEENTH ANNUAL CLINICAL SESSION OF THE AMERICAN COLLEGE OF PHYSICIANS

San Francisco, Calif., April 4-8, 1932

S. Marx White, President
General Sessions

Wm. J. Kerr, General Chairman
Clinical Program

E. R. Loveland, Executive Secretary
133-135 South Thirty-sixth Street,
Philadelphia, Pa.

GENERAL AND HOTEL HEADQUARTERS: PALACE HOTEL, New Montgomery and Market Sts., San Francisco, Calif.

Registration headquarters, information bureau, railroad office, exhibits and the general assembly hall will be located here. This hotel will also be the Official Headquarters. Members and guests are asked to make their reservations promptly.

LIST OF SAN FRANCISCO HOTELS

Name and Location of Hotel	No. of Rooms	RATE PER DAY Single Room 1 Person With Bath	Without Bath	Double Room 2 Persons With Bath	Without Bath	Twin Beds	Extra Person
Palace, Market and New Montgomery	600	3.50 to 7.00		6.00 to 9.00		7.00 to 10.00	2.00
*Alexander Hamilton, 631 O'Farrell St.	500	$3.50 to $5.00		$4.00 to $6.00		$5.00 — $6.00	$1.50
Ambassador, Eddy and Mason Sts.	150	2.50 — 3.00	$1.50 — $2.00	3.50 — 4.00	$2.50	4.00 — 5.00	1.00
Argonaut, 4th and Market Sts.	400	2.00 — 2.50	1.50 — 2.00	3.00 — 3.50	2.00 — 2.50	3.50 — 4.00	1.00
Bellevue, Geary and Taylor Sts.	300	2.50		4.00		5.00	1.50
Brayton, 50 Turk St.	110	1.75 to 2.50	1.25 — 1.50	2.00 to 3.00	1.75 — 2.00	3.00 — 3.50	.50
Californian, 405 Taylor St.	325	3.00 to 4.00		4.00 to 6.00		5.00 — 6.00	1.00
Canterbury, 750 Sutter St.	250	2.50 to 4.50		3.50 to 5.00		4.50 to 6.00	1.00
Cartwright, 524 Sutter St.	130	2.00 to 2.50		2.50 — 3.00		4.00	1.00
Chancellor, 433 Powell St.	150	2.50		3.50		4.00	1.00
Clark, 217 Eddy St.	140	2.00 — 2.50	1.50	2.75 to 3.50	2.50	3.50	1.00
Clift, Geary at Taylor St.	540	3.00 to 5.00		5.00 to 7.00		6.00 to 8.00	2.00
Colonial, 650 Bush St.	150	2.50	1.50	3.00 — 3.50	2.00	4.00	1.00
Continental, 127 Ellis St.	186	2.00	1.50	3.50	2.50	3.50	.50
Court, 555 Bush St.	133	2.00 — 2.50	1.50	3.00 — 3.50	2.00	3.50 — 4.00	1.00
Dale-Tallac, 140 Ellis St.	152	2.00	1.00 up	2.50	1.50 up		.50
Dalt, 34 Turk St.	180	1.50 — 2.00	1.00 — 1.50	2.00 — 2.50	1.50 — 2.00		.50
El Drisco, 2901 Pacific Ave. at Broderick	65	3.00	1.50	4.00	3.50 — 4.50	5.00	1.00
*El Mirasol, 30 Franklin St.	105	1.50 — 2.00	1.00	2.00 — 2.50	1.50		.50
Fairmont, California and Mason Sts.	500	3.50 to 8.00		5.00 to 10.00			2.00
Federal, 1087 Market St.	174	2.00 to 2.50	1.25 1.50	2.50 to 3.00	1.75 — 2.00	2.50 to 3.50	.50
Fielding, Geary and Mason Sts.	105	2.50 — 3.00		3.50 — 4.00		4.00 — 5.00	1.00
Franciscan, 352 Geary St.	175	2.00 — 2.50	1.50	2.50 — 3.00	2.00	4.00	1.00

*Also Hotel Apartments

Name and Location of Hotel	No. of Rooms	RATE PER DAY											
		Single Room 1 Person				Double Room 2 Persons				Twin Beds			Extra Person
		With Bath		Without Bath		With Bath		Without Bath					
Baylord, 620 Jones St.	200	3.50				5.00				6.00			1.50
Benburn, 246 McAllister St.	65	2.00				2.50				3.00			1.00
Golden State, Powell and Ellis Sts.	181	2.00		1.50		3.50		2.50		3.50			.50
Governor, 180 Turk St.	150	2.00	— 2.50			2.50	— 3.00			3.50	—	4.00	1.00
Herbert's Bachelor, 151-159 Powell St.	108	2.00		1.50		3.00		2.50		3.50			1.00
Huntington, 1075 California St.	144	3.50				5.00				5.00	—	6.00	2.00
Keystone, 54 4th St.	168	2.00	to 2.50	1.25	— 1.50	2.50	to 3.00	1.75	— 2.00	3.50			.50
King George, 334 Mason St.	200	2.00				2.50				3.50			.50
Lankershim, 55 - 5th St.	350	2.00	— 2.50	1.25	— 1.50	2.50	— 3.00	1.50	— 2.00	3.00	—	3.50	.50
La Salle, 225 Hyde St.	125	2.00	— 2.50			2.50	— 3.00			3.50	—	4.00	.50
Lombard, 1015 Geary St.	102	2.00	to 2.50			3.00	to 3.50			4.00			1.00
Mark Hopkins, 999 California St.	500	4.00	to 8.00			6.00	to 12.00			7.00	to	14.00	2.00
land, 490 Geary St.	84	1.75		1.50		2.25		2.00		2.75			.50
rice, 761 Post St.	150	3.00	to 4.00			4.00	to 5.00			5.00	to	6.00	1.00
mpic, 230 Eddy St.	250	2.00	— 2.50			3.00	— 3.50			4.00			1.00
nford, Market and Mason Sts.	90	2.00	to 3.00			2.50	to 3.50			4.00			.50
Pickwick, 5th near Market St.	200	2.00	— 2.50			3.00	— 3.50			4.00			1.00
Plaza, Stockton and Post Sts.	300	2.50		2.00		3.50		3.00		4.50	—	5.00	1.00
Powell, 17 Powell St.	205	2.00	— 2.50	1.50		2.50	— 3.00	2.00		3.50			.50
Ramona, 174 Ellis St.	120	2.00				2.50				3.00			.50
Roosevelt, Jones at Eddy St.	200	2.00	— 2.50			2.50	to 3.50			3.50	—	4.00	1.00
St. Andrew, 440 Post St.	60	2.00		1.50		2.50		2.00		3.50	—	4.50	1.00
St. Francis, Powell and Geary Sts.	1000	3.50	to 8.00			5.00	to 9.00			7.00	to	12.00	2.00
Senate, 467 Turk St.	96	2.00		1.50		2.50	— 3.00	2.00		3.00	—	3.50	.50
Senator, 519 Ellis St.	120	2.00	— 2.50			2.50	— 3.00			3.00	—	3.50	.50
Shaw, 1112 Market St.	150	2.50				3.00				3.50			1.00
Sir Francis Drake, Sutter and Powell	600	3.50	to 5.00			5.00	to 7.00			6.00	to	8.00	2.00
Spaulding, 240 O'Farrell St.	132	2.00	— 2.50	1.50	— 2.00	2.50	— 3.00	2.00	— 2.50	4.00			.50
Stewart, 353 Geary St.	400	2.00	to 3.00	1.50	to 2.50	3.50	to 5.00	2.50	— 3.00	4.00	to	5.00	1.00
Stratford, 242 Powell Sts.	110	2.00	— 2.50	1.25	— 1.50	2.50	to 3.00	1.75	— 2.00	3.00	—	3.50	.50
Sutter, Kearny and Sutter Sts.	232	2.00	to 3.00	1.50		2.50	to 3.50	2.00		4.00			.50
Washington, Grant Ave. and Bush St.	200	2.00	to 3.00	1.50	— 2.50	2.50	to 3.50	2.00	— 2.50	4.00			1.00
Whitcomb, Market and 8th Sts.	500	2.50	to 4.00	2.50	to 3.50	4.00	to 6.00	3.00	to 4.00	4.00	to	6.00	1.50
Willard, 161 Ellis St.	125	2.00	— 2.50	1.50		2.50	— 3.00	2.00		3.50	—	4.00	1.00
William Taylor, McAllister and Leavenworth Sts.	500	3.00	to 5.00			4.00	to 7.00			4.00	to	7.00	
Wiltshire, 340 Stockton St.	119	2.00	to 3.00			2.50	to 4.00			4.00			1.00
Worth, 641 Post St.	96	2.00				2.50							1.00

WHO MAY REGISTER—

(a) All members of the American College of Physicians in good standing for 1932.
(b) All newly-elected members.
(c) Members of the San Francisco County Medical Society, without registration fee, upon presentation of their 1932 membership cards in their local society.
(d) Medical students pursuing courses at the University of California Medical School and the Stanford University School of Medicine, upon presentation of matriculation cards or other evidence of registration at these institutions.
(e) House Officers of the hospitals participating in the program.
(f) Members of the Medical Corps of the Public Services of the United States and Canada, without fee, upon presentation of evidence of their appointments.
(g) Qualified physicians who may wish to attend this Session as visitors. Such visiting guests shall pay a registration fee of $15.00, and shall be entitled to one year's subscription to "Annals of Internal Medicine" (in which the proceedings will be published), included within said fee.

REGISTRATION BLANKS FOR ALL SPECIAL CLINICS AND DEMONSTRATIONS are sent to members with the official program. Guests may secure a copy of the program by request to the Executive Secretary.

TRANSPORTATION to and from the San Francisco Clinical Session has been arranged on the Identification Certificate plan of fare and half fare. With the exception of the southeast territory, this reduced rate applies for return by diversified routes. In the southeast territory, members must return by the same as the going route, in order to secure the fare and half fare rate. For diverse return routes in the southeast, the fare will be one and three-fifths of the one way tariff.

Reduced fares apply not only to members, but also to dependent members of their families.

Before purchasing tickets, members must secure from the Executive Secretary an Identification Certificate, to entitle them to the reduced fares.

In general, tickets will be sold from March 26 to April 8, with a return limit of thirty days in addition to date of sale. The fare for children of five, and under twelve, years of age will be one-half of the round trip fare for adults. Children under five years of age free when accompanied by parent or guardian. Stop-overs will be allowed at all stations within final limit on either going or return trip, or both, on application to conductors.

All tickets must be validated by a special railroad agent at the San Francisco headquarters from April 4 to 8.

Official itinerary of special train and post-convention tour. Arrangements have been made for a special train from the East to San Francisco, over the Baltimore & Ohio, Chicago & Northwestern, Union Pacific and Southern Pacific Railroads. Following the Session it will proceed over the Southern Pacific, Santa Fe and Baltimore & Ohio railroads back East, according to the following schedule, which has been arranged for the greater comfort and pleasure of members en route. A special booklet has been distributed to every member.

SCHEDULE

Lv. New York (West 23d St. Station), B. & O. R. R..... 10:32 A.M.—March 31

Lv. New York (42d St. Station), B. & O. R. R..... 10:10 A.M.—March 31
Lv. New York (Liberty St. Station), B. & O. R. R..... 10:48 A.M.—March 31

Brooklyn, Liberty Street Coach Route
Lv. Brooklyn (Joralemon Street Station), B. & O. R. R..... 10:10 A.M.—March 31

Lv. Liberty Street Station, B. & O. R. R.		10:48 A.M.	March 31
Lv. New York (Jersey City Station Capitol Limited)		11:00 A.M.	March 31
Lv. Newark (Motor Coach Connections), B. & O. R. R.		10:55 A.M.	March 31
Lv. Elizabeth, B. & O. R. R.		11:16 A.M.	March 31
Lv. Philadelphia, B. & O. R. R.		12:55 P.M.	March 31
Lv. Wilmington, B. & O. R. R.		1:28 P.M.	March 31
Lv. Baltimore (Mt. Royal Station), B. & O. R. R.		2:48 P.M.	March 31
Lv. Baltimore (Camden Station), B. & O. R. R.		2:55 P.M.	March 31
Lv. Washington, D. C., B. & O. R. R.		4:05 P.M.	March 31
Lv. Cumberland, B. & O. R. R.		7:20 P.M.	March 31
Lv. McKeesport, B. & O. R. R.		10:39 P.M.	March 31
Lv. Pittsburgh (Fort Pitt Limited), B. & O. R. R.		9:30 P.M.	March 31
Lv. Youngstown, B. & O. R. R.		11:42 P.M.	March 31
Lv. Akron, B. & O. R. R.		12:58 A.M.	April 1
Ar. Chicago, B. & O. R. R.	(C.T.)	8:05 A.M.	April 1
Ar. Chicago, B. & O. R. R.	(C.T.)	9:00 A.M.	April 1
Lv. Chicago, C. & N. W. R. R.		10:30 A.M.	April 1
Ar. Omaha, C. & N. W. R. R.		10:30 P.M.	April 1
Lv. St. Louis, Wabash R. R.		7:30 P.M.	March 31
Ar. Omaha, Wabash R. R.		8:00 A.M.	April 1
Lv. Kansas City, Mo. Pac. R. R.		9:00 A.M.	April 1
Ar. Omaha, Mo. Pac. R. R.		3:30 P.M.	April 1
Lv. Kansas City, C. B. & Q. R. R.		12:30 P.M.	April 1
Ar. Omaha, C. B. & Q. R. R.		6:10 P.M.	April 1
Lv. Minneapolis, C. & N. W. R. R.		9:20 A.M.	April 1
Lv. St. Paul, C. & N. W. R. R.		9:55 A.M.	April 1
Lv. Rochester, C. & N. W. R. R.		9:40 A.M.	April 1
Lv. Sioux City, C. & N. W. R. R.		7:15 P.M.	April 1
Ar. Omaha, C. & N. W. R. R.		10:00 P.M.	April 1
Lv. Omaha, Union Pac. R. R.		10:30 P.M.	April 1
Ar. Ogden, Union Pac. R. R.	(M.T.)	11:00 P.M.	April 2
Lv. Ogden, So. Pac. R. R.	(P.T.)	10:15 P.M.	April 2
Ar. Oakland, So. Pac. R. R.		7:00 P.M.	April 3
Ar. San Francisco (by ferry from Oakland)		7:30 P.M.	April 3

POST-CONVENTION TOUR

Lv. San Francisco, So. Pac. R. R.		11:40 P.M.	April 8
Ar. Merced (Yosemite Valley), So. Pac. R. R.		4:55 A.M.	April 9
Note: Saturday, April 9, spent in Yosemite Valley			
Lv. Merced, So. Pac. R. R.		9:35 P.M.	April 9
Lv. Fresno, So. Pac. R. R.		11:15 P.M.	April 9
Ar. Los Angeles, So. Pac. R. R.		8:35 A.M.	April 10
Note: Los Angeles Headquarters—Los Angeles Biltmore Hotel. Program of entertainment in Los Angeles, Sunday and Monday, April 10-11.			
Lv. Los Angeles, Santa Fe R. R.		12:30 P.M.	April 12

Ar. Grand Canyon, Santa Fe R. R.	8:00 A.M.	April 13
Note: Entire day spent at Grand Canyon with headquarters at El Tovar Hotel.		
Lv. Grand Canyon, Santa Fe R. R.	7:45 P.M.	April 13
Ar. Winslow, Santa Fe R. R.	7:40 A.M.	April 14
Note: At this point members may leave the train, and visit the Petrified Forest of Arizona, rejoining the same train by motor coach at Holbrook.		
Ar. Albuquerque, Santa Fe R. R.	4:15 P.M.	April 14
Ar. Lamy, Santa Fe R. R.	6:00 P.M.	April 14
Note: From this point is operated the Indian Detours. Special one, two, or three-day motor tour to the New Mexico Rockies, visiting several Indian pueblos and ancient cliff ruins, may be arranged here.		
Ar. Kansas City, Santa Fe. R. R.	5:15 P.M.	April 15
Note: Members from Minneapolis, St. Paul, Omaha, St. Louis and points in the southeast will leave the party here.		
Lv. Kansas City, Santa Fe R. R.	6:15 P.M.	April 15
Ar. Chicago, Santa Fe R. R.	7:20 A.M.	April 16
Lv. Chicago, B. & O. R. R.	1:45 P.M.	April 16
Ar. Washington, D. C., B. & O. R. R.	8:40 A.M.	April 17
Ar. Baltimore (Camden Station), B. & O. R. R.	9:43 A.M.	April 17
Ar. Baltimore (Mt. Royal Station), B. & O. R. R.	9:51 A.M.	April 17
Ar. Wilmington, B. & O. R. R.	11:11 A.M.	April 17
Ar. Philadelphia, B. & O. R. R.	11:44 A.M.	April 17
Ar. New York (Jersey City Station), B. & O. R. R.	1:43 P.M.	April 17

Those not desiring to take the post-convention tour may return East by regular train over the same or diversified routes from San Francisco, in a period of from four to five days, depending on the route selected. They may, of course, go out with the special train and return by other routes, either railroad, steamship by way of the Panama Canal, or by air. There is a sailing over the Panama Pacific Line from Los Angeles, on April 11, arriving in New York April 25. Full details will be furnished by the Executive Secretary upon request.

Air travel by way of the United Air Lines to the convention may be arranged at the following rates, and in the time indicated.

New York to San Francisco	$160.00	31 hours
Chicago to San Francisco	115.00	23¼ hours
Omaha to San Francisco	95.76	18½ hours
Seattle to San Francisco	43.98	7 hours
Los Angeles to San Francisco	18.95	3 hours

Similar rates from 137 cities in 30 states.

THE GENERAL BUSINESS MEETING OF THE COLLEGE will be held in San Francisco on Thursday, April 7, at 4:30 P.M., immediately following the general scientific program of the afternoon. All Masters and Fellows of the College are urged to be present. There will be the election of Officers, Regents and Governors, the reports of the Treasurer and the Executive Secretary, and the induction to office of the new President, Dr. Francis M. Pottenger, Monrovia, Calif.

THE CONVOCATION OF THE COLLEGE for the induction of newly-elected Fellows will take place Wednesday evening, April 6, at 8:00 P.M., in the Gold Ballroom of the Palace Hotel.

After the induction of the new members, Dr. O. T. Avery, of New York City, will deliver an address on "The Rôle of Specific Carbohydrates in Pneumococcus Infection and Immunity," following which he will be awarded the John Phillips Memorial Prize by the President, Dr. S. Marx White. After this President White will deliver the annual presidential address.

An informal reception to new members will follow immediately after the program.

THE ANNUAL BANQUET OF THE COLLEGE will be held at 7:30 o'clock, Thursday evening, April 7, at the Palace Hotel. All physicians of San Francisco and vicinity and visitors attending the Session are invited, with their ladies, by the members of the College and its Officers to attend this Banquet. A special program will be announced later. Tickets for the Banquet must be purchased at the Registration or Information Bureaus before 10 o'clock, Thursday morning.

ENTERTAINMENT FOR VISITING LADIES. A special program of entertainment for the wives, daughters and friends of attending physicians is being arranged. The ladies are most cordially invited to come to California, and to register with the Ladies' Entertainment Committee.

THE COMMERCIAL EXHIBIT will be located near the Registration Headquarters at the Palace Hotel. The Exhibit will be diversified, consisting of medical literature and texts, pharmaceutical products, apparatus and appliances, special foods, etc. These exhibits are of real scientific value, and every member should definitely arrange to inspect all of them.

FINAL PROGRAM

GENERAL SESSIONS

San Francisco, Calif.—April 4-8, 1932

To provide for the General Sessions a program representative of the best and most advanced work of the year has been more difficult than in some of the previous meetings. The time required for travel by some of the most distant Fellows not as yet air-minded may have proved to be occasionally a deterrent but a survey will show in spite of the difficulties a program of extraordinary breadth and wealth. One circumstance more than compensates for the distances involved for many of the Fellows and Guests. The response to invitations on the part of men in the West and on the Coast has been prompt and satisfactory. A strong and active membership in the West has been faithful and persistent in attendance wherever the College met but, naturally, has been at a numerical disadvantage in the construction of programs. To bring together a large amount of the work of this group provided an opportunity eagerly seized. The large number of new names of active workers is in line with the policies followed in constructing this program. The wealth of material submitted has made selection difficult. The program is an unusually full one.

Special attention may be called to certain features.

1. *Diseases of the kidney* receive consideration in three papers in the opening Session. These papers are practical, of the most lively interest and by men known to every student of the problem.

2. *The liver* is studied from both the experimental and practical standpoint as a part of the program on Tuesday afternoon.

3. On Tuesday evening a strong scientific program will include some of the outstanding work of the *Hooper Foundation* by its director. The work on *pulmonary tuberculosis* will include a summary of the ten years work at the Lymanhurst School for Tuberculous Children, and the relation of the paranasal sinuses to general medicine will be discussed by a long time student of this problem. The *motion picture demonstration* of the factors in the defense of the respiratory mucosa should not be missed.

4. A series of papers on Wednesday afternoon covers some very live topics on the blood vessels and, in addition to papers of practical clinical interest, includes an expo-

sition of the *physics and physiology of arteriosclerosis and hypertension.* New facts about *epilepsy* are discussed and the Director of the Hooper Foundation closes the afternoon with an address on the protective measures of the State of California against *food poisoning.*

5. *The endocrines* do not go unheralded. Sober masters of some of their mysteries will discuss them on Thursday afternoon. For this year the ever-ready heart seems crowded somewhat into the background but *circulatory failure, cardiac complications and the electrocardiogram* are found on the Thursday afternoon program.

6. Symposia have been replaced for the most part by groups of papers as outlined above. One symposium of outstanding significance atones for the numerical lack. The Friday afternoon symposium on the *autonomic nervous system* brings together the most authoritative students of its anatomy, physiology and clinical considerations, together with a résumé of its most recent investigator, the surgeon. It is planned so that the physician may have before him all of the recent great advances in knowledge of this field. It is believed that this symposium alone will make attendance at this Sixteenth Annual Session worth while. The Friday afternoon program closes with three papers of extraordinary interest following the symposium.

The history of medicine is of interest to so large a proportion of the membership that it receives attention again in the General Session. A part of the Monday evening program is given to papers by two delightful exponents of the history of our profession. Medicine in Utopia and the first aphorism of Hippocrates will be treated in a manner no one would wish to miss.

Those interested in the history of medicine will find opportunities in the clinic program unsurpassed at any previous session. The San Francisco Committee has made special provision of papers and exhibits which should call out a large attendance. These will be found in the clinic program.

An intermission has been provided every afternoon except Monday. On that day everyone is fresh and the varied nature of the program makes an intermission less needed. In addition it has been possible to visit the exhibits during the morning and the noon hour. The intermissions will be properly signalled and it is hoped that every member will respond promptly in attending the programs as they are renewed. The program this year is rich and varied and it will be necessary to keep to schedule at every moment.

The importance of visiting the exhibits cannot be overstressed.

OPENING GENERAL SESSION

Monday, April 4, 1932, 2:00 P.M.
Gold Ballroom, Palace Hotel

1. Addresses of Welcome.
   Robert Gordon Sproul, President of the University of California.
   Robert E. Swain, President of Stanford University.
   Langley Porter, Dean of the University of California Medical School.
   William Ophüls, Dean of the Stanford University Medical School.
   Junius B. Harris, President of the California Medical Association.
   Alson R. Kilgore, President of the San Francisco County Medical Society.
2. Reply to Addresses of Welcome.
   S. Marx White,* President of the American College of Physicians.
3. Pathological Differentiations in Bright's Disease.
   Jean Oliver, Brooklyn, N. Y.
   (Guest)
4. Clinical Differentiations in Bright's Disease.
   Thomas Addis,* San Francisco, Calif.

*F. A. C. P.

5. The Relation of Nephrosis to Nephritis.
   E. T. Bell, Minneapolis, Minn.
   (Guest)
6. Clinical Aspects of Gastric Secretion.
   Arthur L. Bloomfield,* San Francisco, Calif.
7. Practical Applications of Recent Discoveries in the Field of Gastro-intestinal Physiology.
   Walter C. Alvarez,* Rochester, Minn.

SECOND GENERAL SESSION
Monday Evening, April 4, 1932, 8:00 P.M.
Gold Ballroom, Palace Hotel
Presiding Officer
Wm. J. Kerr,* San Francisco, Calif.

---

1. The Clinical Study of the Atrophic Tongue.
   Wm. S. Middleton,* Madison, Wis.
2. Medicine in Utopia.
   Richard E. Scammon, Minneapolis, Minn.
   Dean of Medical Sciences of the University of Minnesota.
   (Guest)
3. The First Aphorism of Hippocrates.
   George Dock,* Pasadena, Calif.
4. The Modern Hospital—Its Relationship to the Physician.
   B. W. Black, Oakland, Calif.
   Medical Director of the Highland Hospital of Alameda County.
   (Guest)

THIRD GENERAL SESSION
Tuesday Afternoon, April 5, 1932, 2:00 P.M.
Gold Ballroom, Palace Hotel
Presiding Officer
Walter L. Bierring,* Des Moines, Ia.

---

1. Some Aspects of Bile Function
   Carl L. A. Schmidt, Berkeley, Calif.
   (Guest)
2. The Effect of the Administration of Glucose and Insulin on the Glycogen Content of Normal and Experimentally Damaged Livers.
   T. L. Althausen, San Francisco, Calif.
   (Guest)
3. Further Observations on Primary Carcinoma of the Liver in Chinese.
   G. F. Strong, Vancouver, B. C.
   (Guest)
   H. H. Pitts, Vancouver, B. C.
   (Guest)
4. Hepatic Pathology in Exophthalmic Goiter and the Graves Constitution.
   Carl V. Weller,* Ann Arbor, Mich.
5. The Element of Error in the Diagnosis of Jaundiced Patients—A Review of 500 Cases Verified at Operation or Autopsy.
   George B. Eusterman,* Rochester, Minn.

*F. A. C. P.

6. Asthenia—Clinical Types and Principles of Therapy.
   George Morris Piersol,* Philadelphia, Pa.
   Edward L. Bortz,* Philadelphia, Pa.

INTERMISSION

Visit the Exhibits!

7. The Newer Aspects of Paradentosis (Pyorrhea).
   Hermann Becks, San Francisco, Calif.
   (Guest)
8. The Rôle of Bacteria in Asthma.
   Robert L. Benson, Portland, Ore.
   (Guest)
9. Rocky Mountain Spotted Fever.
   G. Gill Richards,* Salt Lake City, Utah.
10. The Significance of Fever.
    Hobert A. Reimann, Minneapolis, Minn.
    (Guest)
11. Poisonous Spider Bites.
    Emil Bogen, Olive View, Calif.
    (Guest)
12. Studies on the Chemotherapy of Amebiasis.
    Chauncey D. Leake, San Francisco, Calif.
    (Guest)

FOURTH GENERAL SESSION

Tuesday Evening, April 5, 1932, 8:00 P.M.
Gold Ballroom, Palace Hotel
Presiding Officer
J. C. Meakins,* Montreal, Que.

---

1. Recent Studies in Equine Encephalomyelitis. Discussion and Motion Picture Films.
   Karl F. Meyer, San Francisco, Calif.
   Director of the George Williams Hooper Foundation.
   (Guest)
2. Ten Years at the Lymanhurst School for Tuberculous Children.
   J. Arthur Myers,* Minneapolis, Minn.
3. Treatment of Cavities in Pulmonary Tuberculosis.
   LeRoy S. Peters,* Albuquerque, N. M.
4. Atelectasis and Tuberculosis.
   W. Warner Watkins,* Phoenix, Ariz.
   H. P. Mills,* Phoenix, Ariz.
   Fred G. Holmes,* Phoenix, Ariz.
5. The Paranasal Sinus Problem in the Practice of Medicine.
   Arthur D. Dunn,* Omaha, Nebr.
6. Mechanical Factors in the Defense of the Respiratory Mucosa. A Motion Picture Demonstration.
   Anderson Hilding, Rochester, Minn.
   (Guest)

*F. A. C. P.

FIFTH GENERAL SESSION

Wednesday Afternoon, April 6, 1932, 2:00 P.M.
Gold Ballroom, Palace Hotel
Presiding Officer
George Morris Piersol,* Philadelphia, Pa.

1. The Treatment of Raynaud's Disease by Repeated Exposure to Cold.
   Wm. J. Kerr,* San Francisco, Calif.
2. Physics and Physiology of Arteriosclerosis and Hypertension.
   Carl J. Wiggers,* Cleveland, Ohio.
3. Aortic Hypoplasia as a Cause of Death.
   Edgar T. Herrmann,* St. Paul, Minn.
4. Clinical Diagnosis of Pulmonary Arteriosclerosis.
   Henry L. Ulrich,* Minneapolis, Minn.
5. Primary (Essential) Hypertension—A Clinical and Morphological Study of Three Hundred and Seventy-five Cases.
   Francis D. Murphy,* Milwaukee, Wis.
6. Hypertension—A Follow-up Study After Eight to Fifteen Years.
   J. N. Blackford,* Seattle, Wash.

INTERMISSION

Visit the Exhibits!

7. Demonstration and Use of the Radiotherm in Disease of the Circulation.
   C. F. Tenney,* New York, N. Y.
8. Experimental Bases for Vaccine Treatment of Chronic Arthritis with Summary of Results of Treatment.
   B. J. Clawson, Minneapolis, Minn.
   (Guest)
9. Metabolic Abnormalities in Obesity.
   Russell Wilder, Rochester, Minn.
   (Guest)
10. Some Recent Observations Regarding the Nature of Epilepsy.
    Irvine McQuarrie, Minneapolis, Minn.
    (Guest)
11. The Present Status of the Ketogenic Diet in the Treatment of Epilepsy.
    D. Schuyler Pulford,* Woodland, Calif.
12. The Protective Measures of the State of California Against Food Poisoning.
    Karl F. Meyer, San Francisco, Calif.
    Director of the George Williams Hooper Foundation.
    (Guest)

Wednesday Evening, 8:00 P.M.
Gold Ballroom, Palace Hotel

CONVOCATION OF THE COLLEGE

The general profession and the general public are cordially invited. No special admission tickets are required. Evening dress is recommended.

1. Convocation Ceremony.
2. Address: The Rôle of Specific Carbohydrates in Pneumococcus Infection and Immunity.
   O. T. Avery, New York, N. Y.

*F. A. C. P.

3. Presentation of the John Phillips Memorial Prize.
4. Presidential Address.
S. Marx White, Minneapolis, Minn.

Reception to New Members

An informal reception to new members will follow immediately after the Convocation exercises. Newly inducted Fellows should sign the Roster and secure their Fellowship Certificates following the Convocation program.

SIXTH GENERAL SESSION
Thursday Afternoon, April 7, 1932, 2:00 P.M.
Gold Ballroom, Palace Hotel
Presiding Officer
Charles G. Jennings,* Detroit, Mich.

---

1. The Biological and Clinical Importance of Ovary-Stimulating Substances.
C. Frederic Fluhmann, San Francisco, Calif.
(Guest)
2. Animal Experiments with Adrenal Cortical Extracts.
C. L. Connor, San Francisco, Calif.
(Guest)
J. L. Carr, San Francisco, Calif.
(Guest)
3. A Chemical Study of the Suprarenal Gland.
E. C. Kendall, Rochester, Minn.
(Guest)
4. The Clinical Syndromes of Adrenal Hyperfunction. Illustrated by Lantern Slides.
Hans Lisser,* San Francisco, Calif.
5. Various Clinical Syndromes Associated with Diseases of the Suprarenal Glands.
L. R. Rowntree,* Rochester, Minn.

INTERMISSION
Visit the Exhibits!

6. Modern Muscle Physiology and Circulatory Failure.
Jonathan C. Meakins,* Montreal, Que.
7. The Cardiac Complications of Funnel-breast.
James Gray Carr,* Evanston, Ill.
8. A Study of 800 Abnormal Electrocardiograms and Associated Clinical Findings.
Martin A. Mortensen,* Battle Creek, Mich.

The Annual General Business Meeting of the College will be held immediately after the last paper. All Masters and Fellows are urged to be present. Official reports from the Executive Secretary and Treasurer will be read; new Officers, Regents and Governors will be elected, and the President-Elect Dr. Francis M. Pottenger, will be inducted into office.

Thursday Evening, 7:30 P.M.
Palace Hotel
THE ANNUAL BANQUET OF THE COLLEGE

Toastmaster: Dr. Arthur L. Bloomfield,* Professor of Medicine, Stanford University Medical School.
Address: "A Great Country Doctor."
Dr. Charles J. Singer, University of London, London, England.

*F. A. C. P.

FINAL GENERAL SESSION

Friday Afternoon, April 8, 1932, 2:00 P.M.
Gold Ballroom, Palace Hotel
Presiding Officer
Francis M. Pottenger,* Monrovia, Calif.

SYMPOSIUM ON THE AUTONOMIC NERVOUS SYSTEM

1. Anatomy.
   a. General Considerations.
   b. Distribution to Skeletal Muscles and Blood Vessels.
   S. W. Ranson, Chicago, Ill.
   (Guest)
2. The Functions of the Autonomic Nervous System and Its Chemical Agents.
   Walter B. Cannon, Boston, Mass.
   (Guest)
3. Clinical Considerations: Control of Heart, Lungs and Bronchi.
   Harry L. Alexander, St. Louis, Mo.
   (Guest)
4. Control of Blood Pressure and Renal Function.
   Hilding Berglund, Minneapolis, Minn.
   (Guest)

INTERMISSION

Visit the Exhibits!

5. Control of Gastro-intestinal Tract.
   A. J. Carlson,* Chicago, Ill.
6. The Results of Sympathectomy in the Treatment of Peripheral Vascular Diseases, Hirschsprung's Disease and Cord Bladder.
   Alfred W. Adson, Rochester, Minn.
   (Guest)
7. Moving Picture Film "Behaviour of Animals Deprived of the Sympathetic System".
   Walter B. Cannon, Boston, Mass.
   (Guest)
8. Leukopenia: Its Clinical Significance, with Special Reference to Aleukemic Leukemia and Leukemoid Conditions.
   Stacy R. Mettier,* San Francisco, Calif.
9. The Action of Benzol, Roentgen Ray and Radium on the Blood and Blood-forming Organs.
   Edwin E. Osgood, Portland, Ore.
   (Guest)
10. Some Important Factors in Edema Formation.
    Eaton M. MacKay, San Diego, Calif.
    (Guest)

SPECIAL CLINICS AND DEMONSTRATIONS

Clinics and demonstrations will be held in the forenoons from 9:00 to 12:00 daily, Tuesday to Friday, inclusive.

Tickets will be required for each and every one of the special clinics, ward rounds and demonstrations. The co-operation of everyone in securing his clinic tickets will assist greatly in distributing the attendance according to the capacity of each program. It is self-evident that a ward round arranged for twenty-five will lose its value for all if

*F. A. C. P.

forty or fifty are present. Ticket registration naturally is the only effective method of keeping the attendance within the capacities indicated.

To all members of the College, registration blanks for the clinics and demonstrations have been distributed with the final program.

A-I

Tuesday, April 5, 1932

UNIVERSITY OF CALIFORNIA HOSPITAL
(Parnassus and Third Avenues)
Toland Hall, First Floor
(Capacity—167)

9:00- 9:45 Mistakes in the Treatment of Heart Disease.
James B. Herrick, Chicago, Ill.

9:45-10:30 Venous Pressure Determinations as Related to the Diagnosis and Treatment of Cardiac Decompensation.
William S. Middleton, Madison, Wis.

10:30-11:00 Medical Clinic on Cardiovascular Diseases.
William J. Kerr.

11:00-11:30 Medical Clinic on Cardiovascular Diseases.
John J. Sampson.

11:30-12:00 Medical Clinic on Cardiovascular Diseases.
Eugene S. Kilgore.

A-II

UNIVERSITY OF CALIFORNIA HOSPITAL
(Parnassus and Third Avenues)
Room 310, Third Floor
(Capacity—35)
Medical Clinic on Tuberculosis

9:00-10:00 Manifestations of Clinical Tuberculosis in the Adult
F. M. Pottenger, Monrovia, Calif.

10:00-11:00 Juvenile Tuberculosis.
Chesley Bush.

11:00-12:00 Tuberculosis and Tuberculous Infections Among Nurses.
Sidney J. Shipman and Elizabeth A. Davis.

A-III

UNIVERSITY OF CALIFORNIA HOSPITAL
(Parnassus and Third Avenues)
Ward A, Fourth Floor
(Capacity—15)

9:00-10:00 Ward Rounds: Cases of Cholecystitis.
John M. Blackford, Seattle, Wash.

10:00-11:00 Medical Aspects.
Fred H. Kruse.

11:00-12:00 Surgical Aspects.
H. Glenn Bell.

Tuesday, April 5, 1932 (Continued)

A-IV

UNIVERSITY OF CALIFORNIA HOSPITAL
(Parnassus and Third Avenues)
Ward E, Fifth Floor
(Capacity—15)

9:00-10:00 Ward Rounds: Gastro-intestinal Diseases.
George B. Eusterman, Rochester, Minn.

10:00-12:00 Ward Rounds: Gastro-intestinal Diseases.
Elbridge J. Best and Alexander G. Bartlett.

B-I

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
Medical School Building
(Parnassus and Second Avenues)
Cole Hall, Third Floor
(Capacity—200)

9:00- 9:45 The Treatment of Syphilitic Aortitis.
T. Homer Coffen, Portland, Ore.

Symposium on Syphilis:

9:45-11:00 Dermatological Aspects.
Howard Morrow, Hiram E. Miller and Norman N. Epstein.

11:00-12:00 Neurological Aspects.
Edward W. Twitchell.

B-II

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
Medical School Building
(Parnassus and Second Avenues)
Pharmacology Laboratory, Third Floor
(Capacity—50)

9:00- 9:30 Intravenous Magnesium Sulphate in Hypertension.
H. H. Lissner, Los Angeles, Calif.

9:30-10:00 A Study of Substitutes for Epinephrin.
C. H. Thienes, Los Angeles, Calif.

10:00-11:00 Recent Advances in Pharmacology.
Chauncey D. Leake.

11:00-12:00 Discussion and Demonstration of Vaccines and Sera.
Max S. Marshall.

B-III

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
Medical School Building
(Parnassus and Second Avenues)
Medical Teaching Room, First Floor
(Capacity—34)

9:00-10:00 Lesions of the Diaphragm.
J. Homer Woolsey.

10:00-11:00 Studies in Hemochromatosis.
T. L. Althausen.

11:00-12:00 Use of Potassium Acetate and Quinidine in Cardiac Irregularities.
John B. Lagen.

Tuesday, April 5, 1932 (Continued)

B-IV

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL

Medical School Building
(Parnassus and Second Avenues)

Surgical Teaching Room, First Floor
(Capacity—20)

9:00-10:00 The Problem of Diabetes in Hyperthyroidism.
Henry J. John, Cleveland, Ohio.
10:00-10:30 Relationship between Chronic Thyroiditis and Exophthalmic Goiter.
Henry H. Searls.
10:30-11:00 Basal Rate in Toxic Adenoma.
Henry H. Searls.
11:00-12:00 Malignant Goiter.
Robertson Ward.

C-I

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL

College of Dentistry Building
(Parnassus and First Avenues)

Amphitheater, Third Floor
(Capacity—168)

9:00-10:00 Chronic Chlorosis.
Stacy R. Mettier.
Clinic on Hodgkin's Disease:
10:00-11:00 Clinical Aspects.
Ernest H. Falconer.
11:00-12:00 Roentgenological Aspect.
Robert S. Stone.

C-II

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL

College of Dentistry Building
(Parnassus and First Avenues)

Classroom A, Third Floor
(Capacity—82)
Symposium on Hypertension

9:00-10:00 Arterial Hypertension.
C. G. Jennings, Detroit, Mich.
10:00-10:30 Clinical Aspects of Hypertension.
Ernest S. duBray.
10:30-11:00 Experimental Hypertension.
Dudley W. Bennett.
11:00-11:30 Eyeground Changes in Hypertension.
Joseph L. McCool.

Tuesday, April 5, 1932 (Continued)

C-III

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
College of Dentistry Building
(Parnassus and First Avenues)
Classroom B, Third Floor
(Capacity—40)

9:00-10:00 Subject to be announced.
Charles J. Bloom, New Orleans, La.
Introduction of Communicable Diseases on Ships

10:00-11:00 Pneumonia, Meningitis, Ventilation.
Jacob C. Geiger.

11:00-12:00 Clinical Aspects and Treatment of Communicable Diseases.
Alfred C. Reed.

D-I

UNIVERSITY OF CALIFORNIA
Life Sciences Building, Berkeley
Room 2000, Second Floor
(Capacity—560)
Departments of Bacteriology and Protozoology

10:00-10:30 Bacterial Flora of Infected Antra.
T. D. Beckwith and Francis M. Shook.

10:30-11:00 Human Amebiasis.
C. A. Kofoid.

11:00-11:15 Argasive Ticks of the Genus Ornithodorus.
W. B. Herms.

11:15-11:30 An Obstinate Case of Intestinal Myiasis.
W. B. Herms.

D-II

UNIVERSITY OF CALIFORNIA
Life Sciences Building, Berkeley
Room 2003, Second Floor
(Capacity—208)
Departments of Hygiene and Public Health

10:00-10:30 Statistical Pitfalls in Medical Research.
Frank L. Kelly and E. L. Lucia.

10:30-11:00 The Relation of the Degree of Dysmenorrhea to Health Experience and Physical Measurement.
Ruby L. Cunningham.

11:00-11:30 Demonstration of the Kellogg Test for Diphtheria Immunity.
Wilfred H. Kellogg.

D-III

UNIVERSITY OF CALIFORNIA
Life Sciences Building, Berkeley
Room 2503, Second Floor
(Capacity—208)
Departments of Biochemistry, Nutrition and Physiology

10:00-10:30 The Effect of the Calcium and Phosphorus Content of the Diet and of Vitamin D upon Response to Parathyroid Extract Injection.
A. F. Morgan.

Tuesday, April 5, 1932 (Continued)

10:30-10:50 The Relation of Diffusible Calcium to Certain Diseases.
David M. Greenberg.
10:50-11:10 Some Phases of Carbohydrate Metabolism.
I. L. Chaikoff.
11:10-11:30 Some Phases of the Physiology of Blood Formation.
Skerburne F. Cook.

INSTITUTE OF EXPERIMENTAL BIOLOGY

Note: Demonstrations will be held by Professor Herbert M. Evans and Staff in the Anatomy Department, Life Sciences Building.

E-I

STANFORD UNIVERSITY MEDICAL SCHOOL
Medical School Building
(Clay and Webster Streets)
Lane Hall, Second Floor
(Capacity—333)

9:00-10:00 Medical Clinic on Cardiovascular Diseases.
David P. Barr, St. Louis, Mo.
10:00-11.00 Medical Clinic.
Arthur L. Bloomfield.
11:00-12:00 Medical Clinic.
William F. Cheney.

E-II

STANFORD UNIVERSITY HOSPITAL
(Clay and Webster Streets)
Physiotherapy Department, Second Floor
(Capacity—15)

9:00-12:00 Clinic on Physical Therapeutics.
H. L. Langnecker.

E-III

STANFORD UNIVERSITY HOSPITAL
(Clay and Webster Streets)
Operating Amphitheater, Sixth Floor
(Capacity—71)

9:00- 9:45 Studies with the Closed Intestinal Loop.
George E. Burget, Portland, Ore.
9:45-10:30 Treatment of Stenosis of the Esophagus.
John H. Fitzgibbon, Portland, Ore.
10:30-11:00 Anesthesia from the Internist's Standpoint.
Caroline B. Palmer and Staff.
11:00-11:30 Whole Blood Transfusion; Demonstration of Technique.
LeRoy Brooks.
11:30-12:00 Bone Tumors: A General Practice Problem.
Merrill Mensor.

E-IV

STANFORD UNIVERSITY HOSPITAL
Children's Ward, Fourth Floor
(No Program on Tuesday)

Tuesday, April 5, 1932 (Continued)

E-V

STANFORD UNIVERSITY MEDICAL SCHOOL
Medical School Building
(Clay and Webster Streets)
Room 311, Third Floor
(Capacity—112)

9:00- 9:15 The Use of Dessicated Posterior Pituitary in a Case of Diabetes Insipidus.
Harold B. Myers, Portland, Ore.

9:15- 9:30 The Pathology of Hyperthyroidism.
Frank R. Menne, Portland, Ore.

9:30-10:15 Ovary-Stimulating Substances in the Blood of Women.
C. Frederic Fluhmann.

10:15-11:00 Addison's Disease with Tumors of Suprarenals.
Donald A. Carson.

11:00-11:30 Results after Total Thyroidectomy.
Willard E. Kay and Philip K. Gilman.

11:30-12:00 Experimental Hypophysectomy.
Frederick L. Reichert.

F-I

STANFORD UNIVERSITY MEDICAL SCHOOL
Nurses' Home
(2340 Clay Street)
Assembly Room, First Floor
(Capacity—280)

9:00- 9:45 Generalized Tuberculosis.
Allen K. Krause, Tucson, Ariz.

9:45 10:30 Treatment of Pulmonary Cavities.
Charles W. Mills, Tucson, Ariz.

10:30-11:00 A Study of the Bronchial, Pulmonary and Lymphatic Circulation of the Lungs under Various Pathological Conditions, Experimentally Produced.
Emile F. Holman.

11:00-11:30 A Discussion of the Relationship of Upper Respiratory Infection to Acute and Chronic Tuberculous and Pyogenic Pulmonary Disease.
Philip H. Pierson.

11:30-12:00 Infection of Accessory Nasal Sinuses as a Factor in Diseases of the Lungs.
Samuel H. Hurwitz and Edward C. Sewall.

F-II

STANFORD UNIVERSITY MEDICAL SCHOOL
Nurses' Home
(2340 Clay Street)
Room 5, First Floor
(Capacity—74)

9:00- 9:30 (Subject to be announced later.)
Sydney R. Miller, Baltimore, Md.

9:30-10:00 Demonstration of Coccidioidal Granuloma.
Ernest Dickson and Staff.

10:00-10:30 Remarks on Bone Marrow Biopsy Studies.
Harry A. Wyckoff and Loren R. Chandler.

Tuesday, April 5, 1932 (Continued)

10:30-11:00 Studies in the Behavior of a Benign Transplantable Tumor.
Ludwig A. Emge.
11:00-11:20 Diuretics and the Mechanism of Diuresis.
Andrew B. Stockton.
11:20-11:40 Experimental Studies of Epinephrine Substitutes.
Maurice L. Tainter.
11:40-12:00 Protective Action of Colloidal Dyes in Intoxication.
Paul J. Hanzlik.

F-III

STANFORD UNIVERSITY MEDICAL SCHOOL
Nurses' Home
(2340 Clay Street)
Room 4, First Floor
(Capacity—80)

9:00- 9:30 The Heart in Hypothyroidism.
Homer Rush, Portland, Ore.
9:30-10:00 The Three Common Types of Blood Pressure in Vascular Disease.
Willard J. Stone, Pasadena, Calif.
10:00-10:30 Anomalies of the Great Vessels of the Chest.
William Dock.
10:30-11:00 Graphic Records of Gallop Rhythm.
J. K. Lewis.
11:00-11:20 Prediction and Measurement of the Cardiac Silhouette.
Robert R. Newell.
11:20-11:40 Circulatory Changes in the Fundus Oculi.
Hans Barkan.
11:40-12:00 Pitfalls in the Diagnosis of Hydrothorax in Cardiac Decompensation.
Wm. W. Newman.

G

STANFORD UNIVERSITY MEDICAL SCHOOL
Preclinical Departments, Palo Alto
(No Program on Tuesday)

H-I

SAN FRANCISCO HOSPITAL
(22nd Street and Potrero Avenue)
University of California Service
Ward E, First Floor
(Capacity—20)

9:00-10:00 Ward Rounds (Ward E, First Floor).
LeRoy H. Briggs and Staff.
10:00-11:00 Ward Rounds: Cardiovascular Diseases (Ward H, Fourth Floor).
George Morris Piersol, Philadelphia, Pa.

Tuesday, April 5, 1932 (Continued)

H-II

SAN FRANCISCO HOSPITAL
(22nd Street and Potrero Avenue)
University of California Service
Amphitheater, Third Floor
(Capacity—100)

9:00-11:00 Symposium on Pulmonary Tuberculosis.
Harold Brunn, A. Lincoln Brown and Esther Rosencrantz.

11:00-12:00 Medical X-Ray Conference.
Lloyd Bryan and the Medical and Surgical Staffs.

H-III

SAN FRANCISCO HOSPITAL
(22nd Street and Potrero Avenue)
University of California Service
Council Room, First Floor
(Capacity—35)

9:00-10:00 Differential Diagnosis of Bronchogenic Carcinoma.
Marr Bisaillon, Portland, Ore.

10:00-10:15 Exhibition of Anatomical Drawings.
Curle L. Callander.

10:15-10:45 Exhibition of 350 Gall Stones: Discussion of Their Etiology.
Stanley Mentzer.

10:45-11:00 Motion Picture of Operation of Pulmonary Embolectomy.
A. Lincoln Brown.

11:00-12:00 Recognition of Intestinal Obstruction and Some Aspects of Gastro-intestinal Surgery.
George K. Rhodes.

H-IV

SAN FRANCISCO HOSPITAL
(22nd Street and Potrero Avenue)
University of California Service
Ward 30, Fifth Floor
(Capacity—20)

9:00-10:30 Pediatric Clinic: Endocrine Disorders.
Orville Barbour, Peoria, Ill., and Wm. Anthony Reilly.

10:30-12:00 Ward Rounds: Tuberculosis in Infants with Special Reference to Prognosis.
William Anthony Reilly.

H-V

SAN FRANCISCO HOSPITAL
(22nd Street and Potrero Avenue)
University of California Service
Ward C Office, Third Floor
(Capacity—10)

9:00-11:00 Demonstration of Eyegrounds.
Warren D. Horner.

Tuesday, April 5, 1932 (Continued)

I

CHILDREN'S HOSPITAL
(3700 California Street)
Nurses' Home
Recreation Hall, Annex, First Floor
(Capacity—100)

9:00- 9:40 Allergy in Infancy and Childhood.
George Piness, Los Angeles, Calif.

9:40-10:10 Presentation of Allergic Cases.
J. M. Bierman and H. E. Thelander.

10:10-10:40 Rheumatic Endocarditis: Presentation of Cases.
Ann Purdy.

10:40-11:00 Tremendous Enlargements of the Left Auricle in a Patient with Long-standing Mitral Stenosis, Auricular Fibrillation and Adhesive Pericarditis; Report of a Case.
E. Richmond Ware, Los Angeles, Calif.

11:00-11:30 Blood Pictures of Healthy Infants.
Clain F. Gelston.

11:30-12:00 Presentation of Cases.
Myrl M. Morris.

J

FRANKLIN HOSPITAL
(14th and Noe Streets)
(No Program on Tuesday)

K

FRENCH HOSPITAL
(Geary Street and 5th Avenue)
Auditorium, First Floor
(Capacity—250)

9:00- 9:45 Clinic on Postoperative Pulmonary Complications.
Maurice C. Pincoffs, Baltimore, Md.

9:45-10:30 Visualization of Gall Bladder by Plain Film.
Lloyd B. Crow.

10:30-11:15 Diagnosis and Localization of Tumors of the Brain.
E. B. Towne.

11:15-12:00 Acute Contraction of Muscles Due to Trauma and Excessive Venous Hemorrhage in Closed Fractures of the Extremities.
Ethan H. Smith.

L

LAGUNA HONDA HOME
(7th Avenue and Dewey Blvd.)
(No Program on Tuesday)

M

LETTERMAN GENERAL HOSPITAL
United States Presidio
(No Program on Tuesday)

Tuesday, April 5, 1932 (Continued)

N

MARINE HOSPITAL
(14th Avenue and Lake Street)
(No Program on Tuesday)

O

MARY'S HELP HOSPITAL
(145 Guerrero Street)
(No Program on Tuesday)

P-I

MOUNT ZION HOSPITAL
Nursing School Auditorium, First Floor
(2345 Sutter Street)
(Capacity—300)

9:00- 9:30 Diagnosis of Organic Diseases of the Nervous System.
Samuel D. Ingham, Los Angeles, Calif.

9:30-10:00 A Few Interesting Neurological Experiences.
W. F. Beerman.

10:00-10:30 Myasthenia Gravis.
Mervyn H. Hirschfeld.

10:30-11:00 Cultivation of Anterior Poliomyelitis Virus.
Frederick Eberson.

11:00-11:30 Physiology of Dreams.
E. O. Jellinek.

11:30-12:00 Thrombosis of the Anterior Spinal Artery.
Julian M. Wolfsohn.

P-II

MOUNT ZION HOSPITAL
Nursing School Classroom, First Floor
(No Program on Tuesday)

Q

ST. FRANCIS HOSPITAL
(Hyde and Bush Streets)
(No Program on Tuesday)

R

ST. JOSEPH'S HOSPITAL
(Park Hill and Buena Vista Avenues)
(No Program on Tuesday)

S

ST. LUKE'S HOSPITAL
(27th and Valencia Streets)
(No Program on Tuesday)

Tuesday, April 5, 1932 (Continued)

T-I

ST. MARY'S HOSPITAL
(Hayes and Stanyan Streets)
Auditorium, Second Floor
(Capacity—400)

9:00- 9:45 A Clinical Review of Pulmonary Infection.
Donald J. Frick, Los Angeles, Calif.

9:45-10:30 Aortic Hypoplasia.
Edgar T. Herrmann, St. Paul Minn.

10:30-11:15 Carbon Dioxide in the Treatment of Bronchopneumonia of Children.
Randolph G. Flood.

11:15-12:00 Systolic Murmurs: Their Significance Evaluated by Fluoroscopic Examination of the Left Auricle in Oblique Position. Moving Pictures.
Harry Spiro.

T-II

ST. MARY'S HOSPITAL
(Hayes and Stanyan Streets)
Lecture Room No. 1, First Floor
(Capacity—75)

9:00-10:00 The Relationship of the Internist to the Surgeon in the Handling of Surgical Diabetes.
Leander A. Riely, Oklahoma City, Okla.

10:00-11:00 Chylothorax.
Thomas J. Lennon.

11:00-12:00 Presentation of Selected Dermatological Cases.
Harry E. Alderson.

T-III

ST. MARY'S HOSPITAL
(Hayes and Stanyan Streets)
Lecture Room No. 2, Third Floor
(Capacity—50)

9:00-10:30 Muscular Dystrophies: Presentation of Cases.
Milton B. Lennon.

10:30-12:00 Scope and Field of the Private Hospital Laboratory.
Elmer W. Smith.

U

SHRINERS HOSPITAL FOR CRIPPLED CHILDREN
(19th Avenue and Moraga Street)
Clinic Waiting Room, First Floor
(Capacity—75)

9:00-10:00 Ward Rounds.
S. L. Haas.

10:00-12:00 Presentation of Cases:
S. L. Haas.

a. Results of Muscle Transplantation in Paralysis Following Anterior Poliomyelitis.
b. Operative Treatment for Tuberculosis of the Hip.
c. Results after Reduction of Congenital Dislocation of the Hip Joint.

Tuesday, April 5, 1932 (Continued)

V

SOUTHERN PACIFIC HOSPITAL
(Fell and Baker Streets)
Auditorium, Fifth Floor
(Capacity—125)

9:00-10:00 The Recognition and Management of Cardiac Pain.
Louis F. Bishop and Louis F. Bishop, Jr., New York, N. Y.

10:00-10:30 Some Etiological Factors in Precordial Pain.
Bernard Kaufman.

10:30-11:30 The Justification for the Diagnosis of Angina Pectoris.
Philip K. Brown.

11:00-12:00 Cervical Sympathectomy:
a. Dissections of the Cervical Sympathetic.
John D. Humber.
b. Operative Results with Demonstration of Cases.
Walter B. Coffey.

W

SAN FRANCISCO COUNTY MEDICAL SOCIETY BUILDING
(2180 Washington Street)
Meeting Room
(Capacity—100)

Early History of Medicine on the Pacific Coast

9:00- 9:45 Mexico.
Nathan Van Patten

9:45-10:30 Peru.
Jay Randolph Sharpsteen.

10:30-11:15 California.
Henry Harris.

11:15-12:00 Pacific Northwest.
Olof Larsell.

Wednesday, April 6, 1932

A-I

UNIVERSITY OF CALIFORNIA HOSPITAL
(Parnassus and Third Avenues)
Toland Hall, First Floor
(Capacity—167)

9:00- 9:30 Clinical Studies on the Treatment of Amebiasis with Carbarsone.
William M. James, Panama, R. P., Hamilton H. Anderson and Dorothy Koch.

9:30-12:00 Clinic on Amebiasis.
Ernest L. Walker, Alfred C. Reed and Chauncey D. Leake.

A-II

UNIVERSITY OF CALIFORNIA HOSPITAL
(Parnassus and Third Avenues)
Room 310, Third Floor
(Capacity—35)

9:00- 9:45 Hemochromatosis, Subacute Yellow Atrophy of the Liver and Iron Retention.
Adolph Sachs, Omaha, Nebr.

Wednesday, April 6, 1932 (Continued)

9:45-10:00 Studies on Cirrhosis of the Liver.
Frank R. Menne, Portland, Ore.
10:00-11:00 Cirrhosis of the Liver.
Fred H. Kruse and T. J. Althausen.
11:00-12:00 Serum Proteins in the Malnourished.
Frederick Bruckman.

A-III

UNIVERSITY OF CALIFORNIA HOSPITAL
(Parnassus and Third Avenues)
Ward A, Fourth Floor
(Capacity—15)

9:00 10:00 Ward Rounds: Cardiovascular Diseases.
George Dock, Pasadena, Calif.
10:00-11:00 Ward Rounds.
Harry L. Alexander, St. Louis, Mo.
11:00-12:00 Ward Rounds.
Herbert W. Allen.

A-IV

UNIVERSITY OF CALIFORNIA HOSPITAL
(Parnassus and Third Avenues)
Ward E, Fifth Floor
(Capacity—15)

9:00-10:00 Ward Rounds: The Heart in Pregnancy.
S. Marx White, Minneapolis, Minn.
10:00-11:00 Ward Rounds: Heart Disease before and after Middle Life.
George E. Ebright.
11:00-12:00 Ward Rounds.
Felix Cunha.

B-I

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
Medical School Building
(Parnassus and Second Avenues)
Cole Hall, Third Floor
(Capacity—200)

9:00- 9:30 1. Some Organizations of Questionable Etiological Significance Isolated from Stools and Urine Submitted for Examination for Members of the Typhoid-Paratyphoid-Dysentery Group.
2. The Nature of the Slow-Lactose-Fermenting B. Coli Found in Stools and Urine.
Harry J. Sears, Portland, Ore.

The Problem of Undulant Fever in the West

9:30-11:00 Etiology and Epidemiology.
Karl F. Meyer.
11:00-12:00 Clinical Types: Presentation of Cases.
William J. Kerr.

Wednesday, April 6, 1932 (Continued)

B-II

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
Medical School Building
(Parnassus and Second Avenues)
Pharmacology Laboratory, Third Floor
(Capacity—50)

9:00-12:00 Clinico-Pathological Conference: Endocrine Disorders.
L. G. Rowntree, Rochester, Minn., H. Lisser, Frank Hinman, Howard W. Fleming and Charles L. Connor.

B-III

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
Medical School Building
(Parnassus and Second Avenues)
Medical Teaching Room, First Floor
(Capacity—34)

9:00-10:30 Clinic on Allergy in Infancy and Childhood.
George Piness, Los Angeles, Calif., Francis S. Smith and Minnola Stallings.

10:30-11:00 Intestinal Allergy.
Albert H. Rowe.

11:00-11:30 The Rôle of Bacteria in Allergy.
Robert L. Benson, Portland, Ore.

11:30-12:00 Medical Clinic on Allergy.
Irwin C. Schumacher.

B-IV

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
Medical School Building
Surgical Teaching Room, First Floor
(Capacity—20)

9:00-10:00 The Problems Relating to the Use of Thallium in Rodent Control.
Tracy I. Storer, Davis, Calif.

10:00-12:00 Experiences in the Recent Outbreak of Thallium Poisoning in California—
Clinical Features.
H. M. Ginsburg, Fresno, Calif.
Pathological Studies on Fatal Cases.
C. E. Nixon, Fresno, Calif.

C-I

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
College of Dentistry Building
(Parnassus and First Avenues)
Amphitheater, First Floor
(Capacity—168)

9:00- 9:45 The Etiology and Treatment of Abscess of the Lung.
James Alex. Miller, New York, N. Y.

9:45-10:15 Pulmonary Suppuration: Medical Aspects.
Sidney J. Shipman.

Wednesday, April 6, 1932 (Continued)

10:15-11:15 Pulmonary Suppuration: Surgical Aspects.
Harold Brunn.
11:15-11:30 Etiology of Pulmonary Emphysema.
M. Prinzmetal.
11:30-12:00 Postoperative Pulmonary Atelectasis.
William B. Faulkner, Jr.

C-II

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
College of Dentistry Building
(Parnassus and First Avenues)
Classroom A, Third Floor
(Capacity—82)

9:00-10:00 Vaccine Treatment of Chronic Arthritis.
B. J. Clawson, Minneapolis, Minn.
Clinics on Backache.
10:00-10:30 Orthopedic Clinic.
LeRoy Abbott.
10:30-11:00 Medical Clinic.
William L. Bender.
11:00-11:30 Gynecological Clinic.
Alice F. Maxwell.
11:30-12:00 Neurological Clinic.
Richard W. Harvey.

C-III

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
College of Dentistry Building
(Parnassus and First Avenues)
Classroom B, Third Floor
(Capacity—40)

9:00- 9:45 Demonstration Clinic on Polycythemia.
Charles T. Stone, Galveston, Texas.
Medical Clinic on Leukemia.
9:45-10:30 Cell Morphology.
James F. Rinehart.
10:30-11:15 Clinical Aspects.
Stacy R. Mettier.
11:15-12:00 Leukemoid Blood States: Cases.
Ernest H. Falconer and Alfred H. Heald.

D

UNIVERSITY OF CALIFORNIA
Preclinical Departments, Medical School
Life Sciences Building, Berkeley
(No program on Wednesday)

Wednesday, April 6, 1932 (Continued)

E-I

STANFORD UNIVERSITY MEDICAL SCHOOL
Medical School Building
(Clay and Webster Streets)
Lane Hall, Second Floor
(No program on Wednesday)

E-II

STANFORD UNIVERSITY HOSPITAL
(Clay and Webster Streets)
Physiotherapy Department, Second Floor
(No program on Wednesday)

E-III

STANFORD UNIVERSITY HOSPITAL
Operating Amphitheater, Sixth Floor
(No program on Wednesday)

E-IV

STANFORD UNIVERSITY HOSPITAL
(Clay and Webster Streets)
Children's Ward, Fourth Floor
(Capacity—15)

9:00-12:00 Pediatric Ward Rounds: Demonstration of Cases and Discussion of Special Topics.
Harold K. Faber and Staff.

E-V

STANFORD UNIVERSITY MEDICAL SCHOOL
Medical School Building
Room 311, Third Floor
(No program on Wednesday)

F-I

STANFORD UNIVERSITY MEDICAL SCHOOL
Nurses' Home
(2340 Clay Street)
Assembly Room, First Floor
(Capacity—280)

9:00- 9:15 Pathological Differentiation in Bright's Disease.
Jean R. Oliver, Brooklyn, N. Y.

9:15 -9:45 Clinic on Diuretics.
Burrell O. Raulston, Los Angeles, Calif.

9:45-10:30 Clinic on Bright's Disease.
Thomas Addis.

10:30-11:00 Physiological Reaction of Insulin.
Dwight E. Shepardson.

11:00-11:30 A High Fat Modification of Joslin's Diabetic Card.
Horace Gray and Jean Stewart.

11:30-12:00 Disorders of Growths, Illustrated with Lantern Slides.
Horace Gray and L. M. Bayer.

Wednesday, April 6, 1932 (Continued)

F-II

STANFORD UNIVERSITY MEDICAL SCHOOL
Nurses' Home
(2340 Clay Street)
Room 5, First Floor
(Capacity—74)

9:00- 9:30 Gastro-intestinal Diseases.
Walter C. Alvarez, Rochester, Minn.

9:30-10:00 Cholecystography.
Edward Leef.

10:00-10:30 Clinical and Pathological Demonstrations.
Gunther W. Nagel.

10:30-11.00 Diagnosis of Amebiasis.
Herbert Gunn.

11:00-11:30 Jaundice.
Donald A. Carson.

11:30-12:00 Indigestion and Related Problems.
Arthur L. Bloomfield and Associates.

F-III

STANFORD UNIVERSITY MEDICAL SCHOOL
Nurses' Home
(2340 Clay Street)
Room 4, First Floor
(Capacity—80)

9:00- 9:30 Occlusive and Vasomotor Diseases Affecting the Extremities.
George E. Brown, Rochester, Minn.

9:30-10:00 Demonstration of Patients.
Walter F. Schaller and Thomas G. Inman.

10:00-10:30 The 1931 Polyneuritis: Demonstration of Patients.
Julian M. Wolfsohn.

10:30-11:00 Clinical Studies in Epilepsy.
Helen Hopkins-Detrick.

11:00-11:20 Study of Variations in the Roentgenological Appearance of Cerebral Arteries.
Melvin Somers.

11:20-11:40 Anionic Bismuth Therapy in Neurosyphilis.
Henry G. Mehrtens and Pearl S. Pouppirt.

11:40-12:00 Typical and Atypical Cranial Neuralgias.
Frederick L. Reichert.

G

STANFORD UNIVERSITY MEDICAL SCHOOL
Preclinical Departments, Palo Alto
Departments of Anatomy and Physiology
Anatomy Lecture Room
(Capacity—80)

10:00-10:45 The Present Status of the Poliomyelitis Problems: Motion Picture of Experimental Poliomyelitis.
E. W. Schultz.
Room 460, Department of Physiology

Wednesday, April 6, 1932 (Continued)

10:45-11:00 Demonstration of the Effect of Some Brain Lesions on Behavior.
Victor Hall.
Room 460, Department of Physiology
11:00-11:30 The Effects of Protein Diet.
J. R. Slonaker.

H

SAN FRANCISCO HOSPITAL
(22nd Street and Potrero Avenue)
Stanford University Service
Medical Amphitheater, Third Floor, Operating Pavilion
(Capacity—105)

9:00- 9:45 Some Types of Blood Dyscrasia.
John H. Musser, New Orleans, La.
9:45-10:30 Treatment of Addisonian Anemia with Digested Beef.
Garnett Cheney.
10:30-11:15 Skin Reactions to the Application of Ice.
George D. Barnett.
11:15-12:00 Jacksonian Epilepsy.
Julian M. Wolfsohn.

I

CHILDREN'S HOSPITAL
(3700 California Street)
(No Program on Wednesday)

J

FRANKLIN HOSPITAL
(14th and Noe Streets)
(No Program on Wednesday)

K-1

FRENCH HOSPITAL
(Geary Street and 5th Avenue)
Auditorium, First Floor
(Capacity—250)

9:00-10:00 Clinic on Peptic Ulcer.
Clement R. Jones, Pittsburgh, Pa.
10:00-10:30 Peptic Ulcer.
Philip K. Brown.
10:30-11:00 Surgical Aspects of Peptic Ulcer.
John W. Cline.
11:00-11:30 Indications for Surgical Intervention in Peptic and Duodenal Ulcers.
Asa W. Collins.
11:30-12:00 Surgical Emergencies of Peptic Ulcers.
W. W. Washburn.

Wednesday, April 6, 1932 (Continued)

K-II

FRENCH HOSPITAL
(Geary Street and 5th Avenue)
Outpatient Department
(Capacity—50)

9:00-12:00 Clinical Demonstration of Unusual Dermatological Cases.
E. D. Chipman and Jay Jacobs.

L

LAGUNA HONDA HOME
(7th Avenue and Dewey Blvd.)
University of California Service
Sun Room, Ward 4, First Floor
(Capacity—50)

9:00- 9:45 A Comparison of the Treatment of Auricular Fibrillation with Whole Leaf and Glucoside Digitalis.
William D. Stroud, Philadelphia, Pa.

9:45-10:30 The Place of the Chronic Hospital in the Community, with Particular Reference to the Care and Study of So-Called Degenerative Diseases.
Ernest S. duBray.

10:30-11:15 Coccidioides of the Skin.
Norman N. Epstein.

11:15-12:00 Tuberculosis and Ano-Rectal Fistulae.
Montague S. Woolf.

M-I

LETTERMAN GENERAL HOSPITAL
(United States Presidio)
Assembly Hall
(Capacity—100)

9:00-10:00 Clinic on Bright's Disease.
Francis D. Murphy, Milwaukee, Wis.

10:00-11:00 The Common Types of Heart Disease, with Presentation of Cases.
Major W. C. Munly, M. C.

11:00-12:00 The Natural History of the Common Types of Heart Disease.
Major W. C. Munly, M. C.

M-II

LETTERMAN GENERAL HOSPITAL
(United States Presidio)
Ward S-1
(Capacity—60)

9:00-10:30 Selected Neurological Cases.
Lt. Col. T. D. Woodson, M. C.

10:30-12:00 Neurosyphilis: Presentation of Cases.
Major W. D. Mueller, M. C.

Wednesday, April 6, 1932 (Continued)

M-III

LETTERMAN GENERAL HOSPITAL
(United States Presidio)
Ward O-1
(Capacity—15)

9:00-10:30 Peptic Ulcer with Presentation of Cases.
Major D. B. Faust, M. C.

10:30-12:00 Malignant Disease of the Colon: Its Diagnosis and Technique of Proctoscopy (X-Ray Clinic Building).
Major D. B. Faust, M. C.

N

MARINE HOSPITAL
(14th Avenue and Lake Street)
(No Program on Wednesday)

O

MARY'S HELP HOSPITAL
(145 Guerrero Street)
(No Program on Wednesday)

P-I

MOUNT ZION HOSPITAL
(2345 Sutter Street)
Nursing School Auditorium, First Floor
(Capacity—300)

9:00-10:00 Demonstration Clinic: Gastro-intestinal Diseases.
Elmer L. Eggleston, Battle Creek, Mich.

10:00-10:30 Hemorrhage of the Upper Intestinal Tract.
Adolph N. Nahman.

10:30-11:00 The Irritable Colon.
Allan L. Cohn.

11:00-11:30 Malignant Lymphoma.
LeRoy H. Briggs.

11:30-12:00 Pathology of Malignant Lymphoma.
Glanville Y. Rusk.

P-II

MOUNT ZION HOSPITAL
(2345 Sutter Street)
Nursing School Classroom, First Floor
(Capacity—47)

9:00- 9:30 Changes in Mitochondria Induced by Alterations in the Glucose-Glycogen Equilibrium.
E. M. Hall, Los Angeles, Calif., and E. M. MacKay, La Jolla, Calif.

9:30-10:15 Modification of Blood Chemistry Tests.
William G. Mossman.

10:15-11:00 Hinton Test for Syphilis.
Edgar J. Munter.

11:00-11:30 Tuberculin Ointment Adhesive Tape Test.
Ernst Wolff.

11:30-12:00 Blood Serum Colloidal Gold Tests in Poliomyelitis Susceptibility.
Frederick Eberson.

Wednesday, April 6, 1932 (Continued)

Q

ST. FRANCIS HOSPITAL
(1190 Bush Street)
Nurses' Lecture Room, Basement
(Capacity—60)

9:00- 9.30 Analysis of Blood Serum with Newer Methods.
Paul B. Roen, Hollywood, Calif.

9:30- 9:50 Etiology of Duodenal Ulcer.
Emmet Allen.

9:50-10:10 Anomalies of the Gastro-intestinal Tract.
James A. Guilfoil.

10:10-10:30 Duodenal Adhesions in Relation to Pathological Gall Bladder.
C. A. Fogerty.

10:30-11:00 Treatment of Pulmonary Tuberculosis by Extra-pleural Paraffin Filling.
Cabot Brown.

11:00-11:30 Therapeutic Problems in Bronchial Asthma.
Edward Matzger.

11:30-12:00 Paranoiac Trends in Women in the Presenile Period.
Edward W. Twitchell.

R

ST. JOSEPH'S HOSPITAL
(Park Hill and Buena Vista Avenues)
Social Hall, Basement
(Capacity—60)

9:00-10:00 Oxygen in Coronary Heart Disease.
Robert I. Rizer, Minneapolis, Minn.

10:10-11:00 Coronary Thrombosis.
Walter L. Bierring, Des Moines, Iowa.

11:00-12:00 Coronary Thrombosis.
Eugene S. Kilgore.

S-I

ST. LUKE'S HOSPITAL
(27th and Valencia Streets)
Clinic Building, Top Floor
(Capacity—50)

9:00-10:00 Ward Rounds.
James H. Means, Boston, Mass.

10:00-11:00 Usual and Unusual Agranulocytic Pictures.
Harold P. Hill.

S-II

ST. LUKE'S HOSPITAL
(27th and Valencia Streets)
Pope Ward, Basement
(Capacity—50)

10:00-12:00 Ward Rounds: Pediatrics.
George D. Lyman.

Wednesday, April 6, 1932 (Continued)

S-III

ST. LUKE'S HOSPITAL
(27th and Valencia Streets)
X-Ray Department, Basement
(Capacity—25)

9:00-12:00 Radiographic Demonstration.
Howard E. Ruggles and Charles D. Fulmer.

S-IV

ST. LUKE'S HOSPITAL
(27th and Valencia Streets)
Clinical Laboratory, Third Floor
(Capacity—25)

9:00-12:00 Clinical Laboratory Demonstration.
W. Parker Stowe and G. D. Delprat.

T-I

ST. MARY'S HOSPITAL
(Hayes and Stanyan Streets)
Auditorium, Second Floor
(Capacity—400)

9:00-10:00 Treatment of Stenosis of the Esophagus.
John H. Fitzgibbon, Portland, Ore.

10:00-11:00 Congenital Abdominal Bands: A Roentgenological and Clinical Review.
John R. O'Neill.

11:00-12:00 Abnormal Physiology of the Gastro-intestinal Tract.
James A. Guilfoil.

T-II

ST. MARY'S HOSPITAL
(Hayes and Stanyan Streets)
Lecture Room No. 1, Third Floor
(Capacity—75)

9:00- 9:45 Moving Picture Film Demonstrating the Effects of Various Irregularities on Dogs' Hearts.
Carl J. Wiggers, Cleveland, Ohio.

9:45-10:00 Tremendous Enlargement of the Left Auricle in a Patient with Long-Standing Mitral Stenosis, Auricular Fibrillation and Adhesive Pericarditis: Report of a Case.
E. Richmond Ware, Los Angeles, Calif.

10:00-10:45 Arteriosclerosis and Diabetes.
Anthony Diepenbrock.

10:45-11:30 Cardiac Irregularities, Illustrated by Moving Pictures on Living Animals.
Harry Spiro.

T-III

ST. MARY'S HOSPITAL
(Hayes and Stanyan Streets)
Lecture Room No. 2, Third Floor
(No Program on Wednesday)

Wednesday, April 6, 1932 (Continued)

T-IV

ST. MARY'S HOSPITAL
(Hayes and Stanyan Streets)
Children's Ward, Fourth Floor
(Capacity—50)

9:00- 9:30 Intelligence Rating in Juvenile Diabetes.
Howard F. West, Los Angeles, Calif.

9:30-11:30 Pediatric Ward Rounds.
Randolph G. Flood.

11:30-12:00 Radium Treatment: Presentation of Cases.
Monica Donovan.

U

SHRINERS HOSPITAL FOR CRIPPLED CHILDREN
(19th Avenue and Moraga Street)
(No Program on Wednesday)

V

SOUTHERN PACIFIC HOSPITAL
(Fell and Baker Streets)
(No Program on Wednesday)

W

HISTORICAL PROGRAM
(No Program on Wednesday)

Thursday, April 7, 1932

A-I

UNIVERSITY OF CALIFORNIA HOSPITAL
(Parnassus and Third Avenues)
Toland Hall, First Floor
(Capacity—167)

9:00-10:00 Evaluation of Insulin After Ten Years.
Russell Wilder, Rochester, Minn.

10:00-11:00 Results of Ten Years' Experience in the Use of Insulin.
Jonathan Meakins, Montreal, Can.

11:00-11:30 Diabetic Coma: The Use of Insulin in Treatment.
H. Clare Shepardson.

11:30-11:45 Coronary Disease in Diabetes.
Howard W. West, Los Angeles, Calif.

11:45-12:00 Relation of Glycogen Formation to Ketosis.
H. J. Deuel, Los Angeles, Calif.

A-II

UNIVERSITY OF CALIFORNIA HOSPITAL
(Parnassus and Third Avenues)
Room 310, Third Floor
(Capacity—35)

9:00-10:00 Clinic on Epilepsy.
Irvine McQuarrie, Minneapolis, Minn.

10:00-12:00 Metabolic Studies in Children.
Francis S. Smyth and Staff.

Thursday, April 7, 1932 (Continued)

A-III

UNIVERSITY OF CALIFORNIA HOSPITAL
(Parnassus and Third Avenues)
Ward A, Fourth Floor
(Capacity—15)

9:00-10:00 Ward Rounds: Vasomotor Diseases Affecting the Extremities.
George E. Brown, Rochester, Minn.

10:00-11:00 Ward Rounds: Circulatory Diseases of the Extremities.
William J. Kerr.

11:00-12:00 Ward Rounds: Diseases of the Spleen.
S. P. Lucia.

A-IV

UNIVERSITY OF CALIFORNIA HOSPITAL
(Parnassus and Third Avenues)
Ward E, Fifth Floor
(Capacity—15)

9:00-10:30 Ward Rounds: Diseases of the Ductless Glands.
James H. Means, Boston, Mass.

10:30-12:00 Ward Rounds: Diseases of the Ductless Glands.
H. Lisser.

BI

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
Medical School Building
(Parnassus and Second Avenues)
Cole Hall, Third Floor
(Capacity—200)

9:00- 9:30 Studies of the Effect of Feeding Various Types of Thyroid Tissues on Hearts of Rabbits.
Frank R. Menne, Portland, Ore.

9:30-10:00 Experimental and Clinical Observations on the Pathology and Treatment of Exophthalmos.
Howard C. Naffziger.

10:00-10:30 Histogenesis of Neuroglia and Oligodentroglia.
Ottiwell W. Jones.

10:30-12:00 Neurosurgical Clinic.
Howard C. Naffziger and Staff.

B-II

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
Medical School Building
(Parnassus and Second Avenues)
Pharmacology Laboratory, Third Floor
(Capacity—50)

9:00- 9:15 An Experimental Study of Cinchophen Hepatitis.
Harold B. Myers, Portland, Ore.

9:15-10:15 Recent Advances in Pharmacology.
Chauncey D. Leake.

10:15-11:00 Discussion and Demonstration of Vaccines and Sera.
Max S. Marshall.

Thursday, April 7, 1932 (Continued)

11:00-11:30 Treatment of Typhoid Fever with Bacteriophage.
Thos. C. McCleave, Berkeley, Calif.
11:30-12:00 Studies of the Clinical Effects of Bacteriophage.
J. F. Kessel, Los Angeles, Calif.

B-III

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
Medical School Building
(Parnassus and Second Avenues)
Medical Teaching Room, First Floor
(Capacity—34)

9:00-10:00 Demonstration Clinic: Diseases of the Nervous System.
Samuel D. Ingham, Los Angeles, Calif.
10:00-11:00 Myotonia Congenita, Myasthenia Gravis and Familial Periodic Paralysis.
Milton B. Lennon.
11:00-12:00 Hourglass Tumors of the Spinal Cord.
Howard A. Brown.

B-IV

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
Medical School Building
(Parnassus and Second Avenues)
Surgical Teaching Room, First Floor
(Capacity—20)
Medico-Surgical Clinic

9:00-10:00 The Irritable Colon as a Factor in Intestinal Stasis.
Elmer L. Eggleston, Battle Creek, Mich.
10:00-11:00 Diseases of the Colon: Ulcerative Colitis.
Montague S. Woolf.
11:00-12:00 Chronic Colitis.
Fred H. Kruse.

B-V

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
Medical School Building
(Parnassus and Second Avenues)
Pathological Laboratory, Third Floor
(Capacity—15)

9:00- 9:30 A Further Study of a White Family Showing Elliptical Erythrocytes.
Warren C. Hunter, Portland, Ore.
9:30- 9:45 Agranulocytic Syndromes.
Edwin E. Osgood, Portland, Ore.
9:45-10:00 The Reticulocyte Stain.
Edwin E. Osgood, Portland, Ore.
10:00-12:00 Interesting and Unusual Blood Smears: Laboratory Demonstration.
Stacy R. Mettier and James F. Rinehart.

Thursday, April 7, 1932 (Continued)

C-I

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
College of Dentistry Building
(Parnassus and First Avenues)
Amphitheater, Third Floor
(Capacity—168)
Mycotic Infections: Coccidioides, Sporotrichosis, Actinomycosis

9:00-10:30 Etiology and Epidemiology.
Karl F. Meyer and Staff.
10:30-12:00 Clinical Aspects.
Howard Morrow and Staff.

C-II

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
College of Dentistry Building
(Parnassus and First Avenues)
Classroom A, Third Floor
(Capacity—82)

9.00-10:00 The Three Common Types of Blood Pressure in Vascular Disease.
Willard J. Stone, Pasadena, Calif.
10:00-12:00 Evolution of Urinary Excretion with Reference to Renal Function.
I. Excretion of Invertebrates.
Frank Hinman.

C-III

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
College of Dentistry Building
(Parnassus and First Avenues)
Classroom B, Third Floor
(Capacity—40)

9:00- 9:45 Nontuberculous Spinal Arthritis.
W. Paul Holbrook, Tucson, Ariz.
Clinic on Tuberculosis
9:45-10:30 Orthopedic.
Geo. C. Hensel.
10:30-11:15 Urological.
Sidney Olsen.
11:15-12:00 Pulmonary Tuberculosis.
Sidney J. Shipman.

D-I

UNIVERSITY OF CALIFORNIA
Life Sciences Building, Berkeley
Room 2000, Second Floor
(No Program on Thursday)

Thursday, April 7, 1932 (Continued)

D-II

UNIVERSITY OF CALIFORNIA
Life Sciences Building, Berkeley
Room 2003, Second Floor
(Capacity—208)

10:00-10:30 Photosensitization.
Harold F. Blum.

10:30-11:00 Morphine Addiction.
Lawrence E. Detrick.

11:00-11:30 Clinical Aspects of Splenic Physiology.
Eric Ogden.

D-III

UNIVERSITY OF CALIFORNIA
Life Sciences Building, Berkeley
Room 2503, Second Floor
(Capacity—208)

10:00-10:30 The Normal Variation in the Clinically Important Blood Constituents of Women and Their Possible Significance.
Ruth Okey.

10:30-11:00 The Rôle of Vitamin B in the Treatment of Undernutrition in Children.
A. F. Morgan.

11:00-11:30 The Treatment of Carcinoma in Experimental Animals by Low Pressure.
Edward S. Sundstroem.

Note: The following laboratory demonstrations will be held on Thursday morning in the Life Sciences Building:

Demonstration in the Field of Human Amebiasis (Room 5077).
C. A. Kofoid.

Mode of Action of Bacteriophage (Room 3543).
A. P. Krueger.

Demonstration of Low Pressure Treatment of Carcinoma in Experimental Animals (Room 5517).
Edward S. Sundstroem.

No tickets will be required for these demonstrations.

The laboratories of Biochemistry, Physiology, Bacteriology, Household Science and Zoology will be open for inspection.

Institute of Experimental Biology:

Demonstrations will be held by Professor Herbert M. Evans and Staff in the Anatomy Department, Life Sciences Building.

E-I

STANFORD UNIVERSITY MEDICAL SCHOOL
Medical School Building
(Clay and Webster Streets)
Lane Hall, Second Floor
(Capacity—333)

10:00-11:00 Medical Clinic.
Arthur L. Bloomfield.

11:00-12:00 Medical Clinic.
William F. Cheney.

Thursday, April 7, 1932 (Continued)

E-II

STANFORD UNIVERSITY HOSPITAL
(Clay and Webster Streets)
Physiotherapy Department, Second Floor
(Capacity—15)

9:00-12:00 Clinic on Physical Therapeutics.
H. L. Langnecker.

E-III

STANFORD UNIVERSITY HOSPITAL
(Clay and Webster Streets)
Operating Amphitheater, Sixth Floor
(Capacity—71)

9:00- 9:45 Clinic on Postoperative Pulmonary Complications.
Maurice C. Pincoffs, Baltimore, Md.
9:45-10:30 Anesthesia from the Internist's Standpoint.
Caroline B. Palmer and Staff.
10:30-11:15 Whole Blood Transfusion; Demonstration of Technique.
LeRoy Brooks.
11:15-12:00 Bone Tumors: A General Practice Problem.
Merrill Mensor.

E-IV

STANFORD UNIVERSITY HOSPITAL
Children's Ward, Fourth Floor
(No Program on Thursday)

E-V

STANFORD UNIVERSITY MEDICAL SCHOOL
Medical School Building
(Clay and Webster Streets)
Room 311, Third Floor
(Capacity—112)

9:00- 9:30 Treatment of Pernicious Anemia.
Cyrus C. Sturgis, Ann Arbor, Mich.
9:30-10:00 A Study of Glycosuria in Hyperthyroidism.
Blair Holcomb, Portland, Ore.
10:00-10:30 Ovary-Stimulating Substances in the Blood of Women.
C. Frederic Fluhmann.
10:30-11:00 Addison's Disease with Tumors of Suprarenals.
Donald A. Carson.
11:00-11:30 Results After Total Thyroidectomy.
Willard E. Kay and Philip K. Gilman.
11:30-12:00 Experimental Hypophysectomy.
Frederick L. Reichert.

Thursday, April 7, 1932 (Continued)

F-I

STANFORD UNIVERSITY MEDICAL SCHOOL
Nurses' Home
(2340 Clay Street)
Assembly Room, First Floor
(Capacity—280)

9:00- 9:45 The Diagnosis and Treatment of Bronchiectasis.
James Alex. Miller, New York, N. Y.

9:45-10:30 A Study of the Bronchial, Pulmonary and Lymphatic Circulation of the Lungs Under Various Pathological Conditions, Experimentally Produced.
Emile F. Holman.

10:30-11:15 A Discussion of the Relationship of Upper Respiratory Infection to Acute and Chronic, Tuberculous and Pyogenic Pulmonary Disease.
Philip H. Pierson.

11:15-12:00 Infection of the Accessory Nasal Sinuses as a Factor in Diseases of the Lungs.
Samuel H. Hurwitz and Edward Sewall.

F-II

STANFORD UNIVERSITY MEDICAL SCHOOL
Nurses' Home
(2340 Clay Street)
Room 5, First Floor
(Capacity—74)

9:00- 9:30 Rocky Mountain Spotted Fever.
G. Gill Richards, Salt Lake City, Utah.

9:30-10:00 Demonstration of Coccidioidal Granuloma.
Ernest Dickson and Staff.

10:00-10:30 Remarks on Bone Marrow Biopsy Studies.
Harry A. Wyckoff and Loren R. Chandler.

10:30-11:00 Studies in the Behavior of a Benign Transplantable Tumor.
Ludwig A. Emge.

11:00-11:20 Diuretics and tthe Mechanisms of Diuresis.
Andrew B. Stockton.

11:20-11:40 Experimental Studies of Epinephrine Substitutes.
Maurice L. Tainter.

11:40-12:00 Protective Action of Colloidal Dyes on Intoxication.
Paul J. Hanzlik.

F-III

STANFORD UNIVERSITY MEDICAL SCHOOL
Nurses' Home
(2340 Clay Street)
Room 4, First Floor
(Capacity—80)

9:00- 9:30 Oxygen in Coronary Heart Disease.
Robt. I. Rizer, Minneapolis, Minn.

9:30-10:00 Arterial Hypertension.
C. J. Jennings, Detroit, Mich.

10:00-10:30 Anomalies of the Great Vessels of the Chest.
William Dock.

10:30-11:00 Graphic Records of Gallop Rhythm.
J. K. Lewis.

Thursday, April 7, 1932 (Continued)

11:00-11:20 Prediction and Measurement of the Cardiac Silhouette.
Robert R. Newell.

11:20-11:40 Circulatory Changes in the Fundus Oculi.
Hans Barkan.

11:40-12:00 Pitfalls in the Diagnosis of Hydrothorax in Cardiac Decompensation.
Wm. W. Newman.

G

STANFORD UNIVERSITY MEDICAL SCHOOL
Preclinical Departments, Palo Alto
(No Program on Thursday)

II-I

SAN FRANCISCO HOSPITAL
(22nd Street and Potrero Avenue)
University of California Service
Ward I, First Floor
(Capacity—20)

9:00-10:00 Ward Rounds.
LeRoy H. Briggs.

10:00-11:00 Ward Rounds.
Sydney R. Miller, Baltimore, Md.

II-II

SAN FRANCISCO HOSPITAL
(22nd Street and Potrero Avenue)
University of California Service
Amphitheater, Third Floor
(Capacity—100)

9:00- 9:30 Pulmonary Emphysema.
Frank R. Mount, Oregon City, Ore.

9:30-10:00 The Paranasal Sinus Problem in Internal Medicine.
A. D. Dunn, Omaha, Nebr.

10:00-10:30 Atelectasis, Clinical and Experimental.
H. W. Stephens and M. Printzmetal.

10:30-11:00 Lesions of the Esophagus.
F. D. Heegler.

11:00-12:00 Medico-Pathological Conference.
Glanville Y. Rusk and Medical and Surgical Staffs.

II-III

SAN FRANCISCO HOSPITAL
(22nd Street and Potrero Avenue)
University of California Service
Council Room, First Floor
(Capacity—35)

The Exhibition of Gall Stones and Exhibition of Anatomical Drawings will be still on display.

Thursday, April 7, 1932 (Continued)

H-H'

SAN FRANCISCO HOSPITAL.
(22nd Street and Potrero Avenue)
University of California Service
Ward 30, Fifth Floor
(Capacity—20)

9:00-10:00 Ward Rounds: Tuberculosis in Children.
J. Arthur Myers, Minneapolis, Minn.

10:00-11:30 Ward Rounds: Tuberculosis in Infants, with Special Reference to Prognosis.
Wm. Anthony Reilly.

11:30-12:00 Intravenous Urography in Children.
Amos U. Christie.

I

CHIDREN'S HOSPITAL.
(3700 California Street)
Recreation Hall, Annex, First Floor
(Capacity—100)
Communicable Disease Department

9:00- 9:45 Use of Spencer-Parker Vaccine in Rocky Mountain Spotted Fever.
R. R. Parker, Hamilton, Mont.

9:45-10:30 Meningococcus Infections.
E. B. Shaw and H. E. Thelander.

10:30-11:15 Blood Picture in Treated, Untreated and in Complicated Pertussis.
Hildegard Henderson and H. E. Thelander.

11:15-12:00 Blood Grouping in Infectious Diseases.
E. B. Shaw and Kathleen Kilgariff.

J

FRANKLIN HOSPITAL.
(14th and Noe Streets)
Nurses' Lecture Room, Second Floor
(Capacity—30)

9:00-10:00 Clinic on Rheumatic Heart Disease and Subacute Bacterial Endocarditis.
Geo. Morris Piersol, Philadelphia, Pa.

10:00-10:30 Clinic on Degenerative Circulatory Diseases.
Emmett C. Taylor.

10:30-11:00 Dietary Principles as Related to Particular Intestinal Conditions.
Elbridge J. Best.

11:00-11:30 Clinic on Interesting Dermatoses.
Harry E. Alderson and Stuart C. Way.

11:30-12:00 Diagnosis and Treatment of Chronic Epidemic Encephalitis.
Walter F. Schaller.

K

FRENCH HOSPITAL.
(Geary Street and 5th Avenue)
Auditorium, First Floor
(Capacity—250)

9:00- 9:45 The Treatment of Syphilitic Aortitis.
T. Homer Coffen, Portland, Ore.

Thursday, April 7, 1932 (Continued)

9:45-10:30 Heart Block.
Eugene S. Kilgore.

10:30-11:15 A Presentation of Cases and Moving Pictures of Auricular Fibrillation and a Discussion of Treatment.
Harry Spiro and Wm. W. Newman.

11:15-11:45 Diseases of the Thyroid Gland: Medical and Surgical Aspects.
Carl L. Hoag.

11:45-12:00 Discussion of Diseases of the Thyroid Gland.
W. W. Washburn.

L

LAGUNA HONDA HOME
(7th Avenue and Dewey Blvd.)
Stanford University Service
Chapel, First Floor, Infirmary Building
(Capacity—300)

9:00-10:00 Differential Diagnosis of Bronchogenic Carcinoma.
Marr Bisaillon, Portland, Ore.

10:00-11:00 Demonstration of X-Ray Films.
Edward Leef.

11:15-12:00 Problems in Hematology.
Garnett Cheney.

M

LETTERMAN GENERAL HOSPITAL
(United States Presidio)
(No Program on Thursday)

N

MARINE HOSPITAL
(14th Avenue and Lake Street)
Board Room No. 100, First Floor
(Capacity—20)

9:00- 9:30 Clinical Studies on the Treatment of Amebiasis with Carbarsone.
Wm. M. James, Panama, R. P., Hamilton H. Anderson and Dorothy Koch

9:30-10:00 Gastric and Duodenal Ulcers.
Robert A. Jones.

10:00-10:30 Malaria.
Walter P. Griffey.

10:30-11:00 Amebiasis.
Richey L. Waugh.

11:00-11:30 Paresis.
Elmer A. Carberry.

11:30-12:00 Arteriosclerosis.
James F. Worley.

Thursday, April 7, 1932 (Continued)

O

MARY'S HELP HOSPITAL
(145 Guerrero Street)
Lecture Room, First Floor
(Capacity—35)

9:00-10:00 Neutrophilic Leukopenia: Report of a Case.
Andrew Bonthius, Pasadena, Calif.

10:00-10:30 Allergy in Children.
Crawford Bost.

10:30-11:00 Medical Aspects of the Toxemias of Pregnancy.
Hans von Geldern.

11:00-11:30 Malignant Neutropenia.
Frank E. Stiles.

11:30-12:00 Intractable Diarrhea.
Alfred C. Reed.

P-I

MOUNT ZION HOSPITAL
Nursing School Auditorium, First Floor
(2345 Sutter Street)
(Capacity—300)

9:00-10:00 A Treatment of Rheumatic Cardiovascular Disease with Special Reference to Intravenous Vaccine.
Wm. D. Stroud, Philadelphia, Pa.

10:00-10:30 Neuromata of the Appendix.
Franklin I. Harris and Morris J. Groper.

10:30-11:00 Perinephric Abscess.
Harold Brunn.

11:00-11:30 Autotransfusion from the Pleura.
A. Lincoln Brown.

11:30-12:00 Accidental Electrical Shock.
Felix Pearl.

P-II

MOUNT ZION HOSPITAL
(2345 Sutter Street)
Nursing School Classroom, First Floor
(Capacity—47)

9:00- 9:30 The Significance of Fever.
Hobart A. Reimann, Minneapolis, Minn.

9:30-10:00 Demonstration of the Use of Radiothermy in Diseases of the Circulation.
C. F. Tenney, New York, N. Y.

10:00-10:30 Pyrodiathermy.
Lloyd Bryan.

10:30-11:00 Medical Aspects of Diseases of the Prostate.
L. C. Jacobs.

11:00-11:30 Relief of Paralytic Ileus by Spinal Anesthesia.
E. H. Bolze.

11:30-12:00 Immune Rabbit Serum in Staphylococcic Septicemia.
Morris J. Groper.

Thursday, April 7, 1932 (Continued)

ST. FRANCIS HOSPITAL
(1190 Bush St.)
(No Program on Thursday)

R

ST. JOSEPH'S HOSPITAL
(Park Hill and Buena Vista Avenues)
(No Program on Thursday)

S

ST. LUKE'S HOSPITAL
(27th and Valencia Streets)
(No Program on Thursday)

T

ST. MARY'S HOSPITAL
(Hayes and Stanyan Streets)
(No Program on Thursday)

U

SHRINERS HOSPITAL FOR CRIPPLED CHILDREN
(19th Avenue and Moraga Street)
(No Program on Thursday)

V

SOUTHERN PACIFIC HOSPITAL
(Fell and Baker Streets)
Auditorium, Fifth Floor
(Capacity—125)

9:00- 9:30 Demonstration Clinic: Gastro-intestinal Diseases.
John Dudley Dunham, Columbus, Ohio.

9:30-10:00 Bidirectional Ventricular Tachycardia Apparently Due to Digitalis.
Wm. H. Leake, Los Angeles, Calif.

10:00-10:30 Management of Peptic Ulcer Among Railroad Employees.
Philip K. Brown.

10:30-11:00 The Place of Laboratory Examinations in the Treatment of Diabetes.
Emmett Allen.

11:00-12:00 Ward Rounds.
Philip K. Brown and Staff.

W

HISTORICAL PROGRAM
(No Program on Thursday)

A-I

Friday, April 8, 1932

UNIVERSITY OF CALIFORNIA HOSPITAL
(Parnassus and Third Avenues)
Toland Hall, First Floor
(Capacity—167)

9:00-11:00 Rocky Mountain Spotted Fever.
R. R. Parker, Hamilton, Mont., G. Gill Richards, Salt Lake City, Utah, and Ernest L. Walker.

11:00-12:00 Relapsing Fever.
Karl F. Meyer.

A-II

UNIVERSITY OF CALIFORNIA HOSPITAL
(Parnassus and Third Avenues)
Room 310, Third Floor
(Capacity—35)

9:00-10:00 Chemistry of Intestinal Obstruction.
Francis S. Smith.

10:00-11:00 Certain Experimental Studies in Intestinal Obstruction.
M. Laurence Montgomery.

11:00-12:00 Treatment of Intestinal Obstruction.
Robertson Ward.

A-III

UNIVERSITY OF CALIFORNIA HOSPITAL
(Parnassus and Third Avenues)
Ward A, Fourth Floor
(Capacity—15)

9:00-10:00 Ward Rounds: Diseases of Blood Formation.
J. H. Musser, New Orleans, La.

10:00-12:00 Ward Rounds.
Ernest H. Falconer and Stacy R. Mettier.

A-IV

UNIVERSITY OF CALIFORNIA HOSPITAL
(Parnassus and Third Avenues)
Ward E, Fifth Floor
(Capacity—15)

9:00-10:00 Ward Rounds.
Leander A. Riely, Oklahoma City, Okla.

10:00-11:00 Ward Rounds: An Instance of Lymphomatosis Radiologically Cured.
Henry Harris

11:00-12:00 Ward Rounds.
Irwin C. Schumacher.

B-I

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
Medical School Building
(Parnassus and Second Avenues)
Cole Hall, Third Floor
(Capacity—200)

9:00-10:00 Peptic Ulcer.
Clement R. Jones, Pittsburgh, Pa.

Friday, April 8, 1932 (Continued)

10:00-11:00 Unusual Gastric Lesions.
J. Homer Woolsey and Fred H. Kruse.
11:00-12:00 X-Ray Therapy of Gastric Lesions.
Howard E. Ruggles.

B-II

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
Medical School Building
(Parnassus and Second Avenues)
Pharmacology Laboratory, Third Floor
(Capacity—50)

9:00-12:00 Clinico-Pathological Conference: Cardiovascular Diseases.
Wm. S. Middleton, Madison, Wis., Henry L. Ulrich, Minneapolis, Minn., Wm. J. Kerr, Gordon E. Hein and Charles L. Connor.

B-III

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
Medical School Building
(Parnassus and Second Avenues)
Medical Teaching Room, First Floor
(Capacity—34)

9:00-10:00 Studies in Psychopathic Personalities.
Hermann M. Adler,
10:00-11:00 Demonstration of Nerve Cases.
Richard W. Harvey.
11:00-12:00 Neurological Aspects of Pernicious Anemia.
Mervyn H. Hirschfeld.

B-IV

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
Medical School Building
(Parnassus and Second Avenues)
Surgical Teaching Room, First Floor
(Capacity—20)

9:00- 9:45 Diverticulosis of the Colon in Relation to Chronic Arthritis.
Ernest C. Fishbaugh, Los Angeles, Calif.
9:45-10:30 Polyposis of the Large Bowel.
Montague S. Woolf.
10:30-12:00 Bone Tumors: Diagnosis and Treatment.
Edwin I. Bartlett and Robert S. Stone.

C-I

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
College of Dentistry Building
(Parnassus and First Avenues)
Amphitheater, Third Floor
(Capacity—168)
Medical Clinic on Endocrinology

9:00-10:00 Demonstration of Cases.
David P. Barr, St. Louis, Mo.

Friday, April 8, 1932 (Continued)

10:00-11:00 Myxedema.
H. Lisser.
11:00-12:00 Diabetes.
H. Clare Shepardson.

C-II

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
College of Dentistry Building
(Parnassus and First Avenues)
Classroom A, Third Floor
(Capacity—82)

9:00- 9:30 Experimental Studies of the Effects of Potassium Bichromate on the Monkey's Kidney.
Warren C. Hunter, Portland, Ore.
9:30-10:00 Glomerular Changes in the Kidneys of Rabbits and Monkeys Produced by Uranium Nitrate, Mercuric Chloride and Potassium Bichromate.
Warren C. Hunter, Portland, Ore.
10:00-12:00 Evolution of Urinary Excretion with Reference to Renal Function.
II. Excretion of Vertebrates.
Frank Hinman.

C-III

UNIVERSITY OF CALIFORNIA MEDICAL SCHOOL
College of Dentistry Building
(Parnassus and First Avenues)
Classroom B, Third Floor
(Capacity—40)

9:00- 9:45 Treatment of Pernicious Anemia.
Cyrus C. Sturgis, Ann Arbor, Mich.
9:45-10:30 Chemotherapy of Amebiasis.
Hamilton H. Anderson.
10:30-11:15 New Drugs in Cardiac Disease.
Dudley W. Bennett.
11:15-12:00 New Drugs Used in Diagnosis.
Chauncey D. Leake.

D

UNIVERSITY OF CALIFORNIA
Preclinical Departments, Medical School
Life Sciences Building, Berkeley
(No Program on Friday)

E-I

STANFORD UNIVERSITY MEDICAL SCHOOL
Medical School Building
(Clay and Webster Streets)
Lane Hall, Second Floor
(No Program on Friday)

Friday, April 8, 1932 (Continued)

E-II

STANFORD UNIVERSITY HOSPITAL
(Clay and Webster Streets)
Physiotherapy Department, Second Floor
(No Program on Friday)

E-III

STANFORD UNIVERSITY HOSPITAL
Operating Amphitheater, Sixth Floor
(No Program on Friday)

E-IV

STANFORD UNIVERSITY HOSPITAL
(Clay and Webster Streets)
Children's Ward, Fourth Floor
(Capacity—15)

9:00-12:00 Pediatric Ward Rounds: Demonstration of Cases and Discussion of Special Topics.
Harold K. Faber and Staff.

E-V

STANFORD UNIVERSITY MEDICAL SCHOOL
Medical School Building
Room 311, Third Floor
(No Program on Friday)

F-I

STANFORD UNIVERSITY MEDICAL SCHOOL
Nurses' Home
(2340 Clay Street)
Assembly Room, First Floor
(Capacity—280)

9:00- 9:45 Clinic on Bright's Disease.
Thomas Addis.

9:45-10:30 Nephritis and Nephrosis.
E. T. Bell, Minneapolis, Minn.

10:30-11:00 Physiological Reaction of Insulin.
Dwight E. Shepardson.

11:00-11:30 High Fat Modification of Joslin's Diabetic Card.
Horace Gray and Jean Stewart.

11:30-12:00 Disorders of Growth, Illustrated with Lantern Slides.
Horace Gray and L. M. Bayer.

F-II

STANFORD UNIVERSITY MEDICAL SCHOOL
Nurses' Home
(2340 Clay Street)
Room 5, First Floor
(Capacity—74)

9:00- 9:30 Prognosis for Cholecystitis, Based on a Clinical Study.
John M. Blackford, Seattle, Wash.

Friday, April 8, 1932 (Continued)

9:30-10:00 Cholecystography.
Edward Leef.

10:00-10:30 Clinical and Pathological Demonstrations.
Gunther W. Nagel.

10:30-11:00 Diagnosis of Amebiasis.
Herbert Gunn.

11:00-11:30 Jaundice.
Donald A. Carson.

11:30-12:00 Indigestion and Related Problems.
Arthur L. Bloomfield and Associates.

F-III

STANFORD UNIVERSITY MEDICAL SCHOOL.
Nurses' Home
(2340 Clay Street)
Room 4, First Floor
(Capacity—80)

9:00- 9:30 Cerebral Vascular Syndromes.
C. J. Rohwer, Seattle, Wash.

9:30-10:00 Demonstration of Patients.
Walter F. Schaller and Thomas S. Inman.

10:00-10:30 The 1931 Polyneuritis: Demonstration of Patients.
Julian M. Wolfsohn.

10:30-11:00 Clinical Studies in Epilepsy.
Helen Hopkins-Detrick.

11:00-11:20 Study of Variations in the Roentgenological Appearance of Cerebral Arteries.
Melvin Somers.

11:20-11:40 Anionic Bismuth Therapy in Neurosyphilis.
Henry G. Mehrtens and Pearl S. Pouppirt.

11:40-12:00 Typical and Atypical Cranial Neuralgias.
Frederick L. Reichert.

G

STANFORD UNIVERSITY MEDICAL SCHOOL
Preclinical Departments, Palo Alto
Anatomy Lecture Room
(Capacity—80)
Department of Anatomy

10:00-10:15 Modification of the Uterine Vascular Rhythms by Certain Pharmacological Agents.
J. E. Markee.

10:15-10:30 Neurologic Mechanism in Spinal Standing in the Cat.
J. C. Hinsey.

10:30-10:45 Hereditary Alopecia Anemia and Duplication of Parts in Some Laboratory Animals.
C. H. Danforth.

10:45-11:30 Bacteriophage as a Therapeutic Agent: A Summary of Clinical Reports Made to the Department of Bacteriology.
E. W. Schultz.

Friday, April 8, 1932 (Continued)

H

SAN FRANCISCO HOSPITAL
(22nd Street and Potrero Avenue)
Stanford University Service
(Medical Amphitheater, Third Floor, Operating Pavilion)
(Capacity—105)

9:00-10:00 Moving Picture Film Demonstrating the Effects of Various Irregularities on Dogs' Hearts.
Carl J. Wiggers, Cleveland, Ohio.

10:00-10:30 Cardiac Asthma.
Charles E. Watts, Seattle, Wash.

10:30-11:00 Bronchial Asthma.
Edward Matzger.

11:00-11:30 Clinical Studies of Circulatory Adaptations.
J. Marion Read.

11:30-12:00 Ward Rounds.
Harold P. Hill.

I

CHILDREN'S HOSPITAL
(3700 California Street)
(No Program on Friday)

J

FRANKLIN HOSPITAL
(14th and Noe Streets)
(No Program on Friday)

K

FRENCH HOSPITAL
(Geary Street and 5th Avenue)
Auditorium, First Floor
(Capacity—250)

9:00- 9:30 Treatment of Typhoid Fever with Bacteriophage.
Thomas C. McCleave, Berkeley, Calif.

9:30-10:00 Report of a Case of Colon Bacillus Meningitis Associated with an Unusual Spinal Anomaly.
Bradford F. Dearing.

10:00-10:30 Laboratory Demonstrations:
1. Easy Measurements of Red Blood Cell Diameter in Pernicious Anemia by Eve's Halometer.
2. Urine Pregnancy Test by Friedman Modification of the Aschheim-Zondek Test.
Marion H. Lippman.

10:30-11:15 Migrating Pneumonia: A Study of Certain Factors which Determine Sites of Migration; Newer Principles of Treatment.
William B. Faulkner, Jr., and Aime N. Fregeau.

11:15-12:00 Middle Lobe Pulmonary Abscesses: Problems in Diagnosis and Treatment.
William B. Faulkner, Jr., and Philip G. Corliss.

Friday, April 8, 1932 (Continued)

L

LAGUNA HONDA HOME
(7th Avenue and Dewey Blvd.)
(No Program on Friday)

M-I

LETTERMAN GENERAL HOSPITAL
(United States Presidio)
Assembly Room
(Capacity—100)

9:00-10:00 Hematoporphyrinuria.
Verne Mason, Los Angeles, Calif.

10:00-11:00 Diabetes Mellitus.
Major D. B. Faust, M. C.

11:00-12:00 Types of Jaundice: Differential Diagnosis and Treatment.
Major D. B. Faust, M. C.

M-II

LETTERMAN GENERAL HOSPITAL
(United States Presidio)
Ward S-1
(Capacity—60)

9:00-10:30 Dementia Praecox, with Presentation of Cases.
Lt. Col. T. D. Woodson, M. C.

10:30-12:00 Selected Psychiatric Cases.
Major T. L. Long, M. C.

M-III

LETTERMAN GENERAL HOSPITAL
(United States Presidio)
Medical Wards
(Capacity—15)

9:00-10:00 Ward Rounds.
James G. Carr, Chicago, Ill.

10:00-11:00 Ward Rounds: Cardiac Section.
Major W. C. Munly, M. C.

11:00-12:00 Ward Rounds: Selected Medical Cases.
Col. L. L. Smith, M. C.

N

MARINE HOSPITAL
(14th Avenue and Lake Street)
(No Program on Friday)

O

MARY'S HELP HOSPITAL
(145 Guerrero Street)
Lecture Room, First Floor
(Capacity—35)

9:00- 9:30 Nontuberculous Spinal Arthritis.
W. Paul Holbrook, Tucson, Ariz.

Friday, April 8, 1932 (Continued)

9:30-10:00 Studies of the Relationship of Dental Infections to So-Called Focal Infections.
Frank R. Menne and Miriam Luetin, Portland, Ore.

10:00-10:30 Pathological Types of Arthritis.
Zera E. Bolin.

10:30-11:00 Arthritis of the Menopause.
J. Morrille George.

11:00-11:30 Studies in the Etiology of Arthritis.
Leon Parker.

11:30-12:00 Treatment of Arthritis.
Merrill C. Mensor.

P-I

MOUNT ZION HOSPITAL
(2345 Sutter Street)
Nursing School Auditorium, First Floor
(Capacity—300)

9:00- 9:30 Diseases of the Gastro-intestinal Tract.
Walter C. Alvarez, Rochester, Minn.

9:30-10:00 Luminal Dermatitis.
Normal N. Epstein.

10:00-10:30 Diabetes Insipidus.
Russell F. Rypins.

10:30-11:00 Mechanical Influence on Clinical Heart Function of Pericardial Lesions.
John J. Sampson.

11:00-11:30 The Severe Chronic Intractable Type of Bronchial Asthma; Reason for Failure.
Fred Firestone.

11:30-12:00 Exudative Lesions in Pulmonary Tuberculosis.
William C. Voorsanger.

P-II

MOUNT ZION HOSPITAL
Nursing School Auditorium, First Floor
(No Program on Friday)

Q

ST. FRANCIS HOSPITAL
(1190 Bush Street)
Nurses' Lecture Room, Basement
(Capacity—60)

9:00- 9:30 The Heart in Hypothyroidism.
Homer Rush, Portland, Ore.

9:30-10:00 Angina Pectoris.
Bernard Kaufman.

10:00-10:30 A Demonstration and Discussion of the Pathology of Coronary Disease.
A. M. Moody.

10:30-11:00 Unusual Blood Reactions to Infection.
Philip K. Brown.

Friday, April 8, 1932 (Continued)

11:00-11:30 Atypical Blood Conditions in Children.
William P. Lucas.

11:30-12:00 A Physiological Comparison of High and Low Carbohydrate Feeding in Diabetes.
Dwight Ervin.

R

ST. JOSEPH'S HOSPITAL
(Park Hill and Buena Vista Avenues)
(No Program on Friday)

S

ST. LUKE'S HOSPITAL
(27th and Valencia Streets)
(No Program on Friday)

T

ST. MARY'S HOSPITAL
(Hayes and Stanyan Streets)
(No Program on Friday)

U

SHRINERS HOSPITAL FOR CRIPPLED CHILDREN
(19th Avenue and Moraga Street)
(No Program on Friday)

V

SOUTHERN PACIFIC HOSPITAL
(Fell and Baker Streets)
(No Program on Friday)

W

LANE LIBRARY
(Clay and Webster Streets)
(Capacity—100)

9:00-12:00 Anatomical Traditions of the Renaissance and Their Relation to the Modern Teaching of Anatomy.
Charles J. Singer, London, Eng., Sanford V. Larkey, John de C. M. Saunders, and others.